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THE TEST-DIET
IN
NTESTINAL DISEASES

PROF. DR. ADOLF SCHMIDT

CHARLES D. AARON, M.D.

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TRITURATED FECES

Plate I

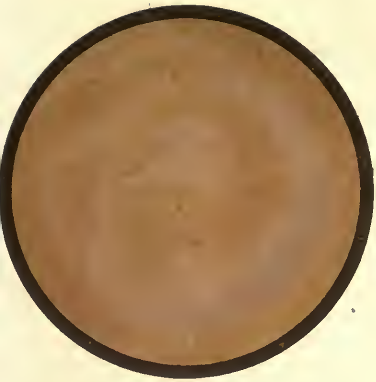

Fig. 1. Normal Feces

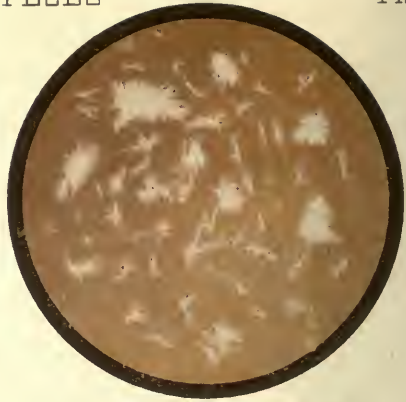

Fig. 2. Connective Tissue Remains

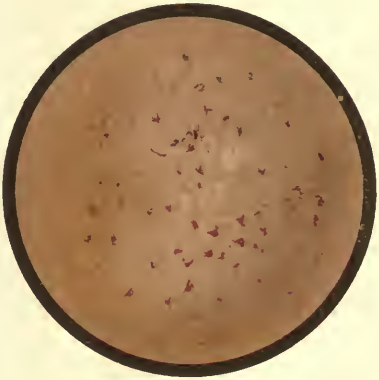

Fig. 3. Muscle Remains

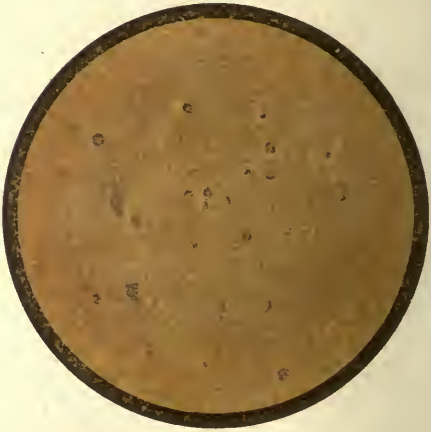

Fig. 4. Potato Remains

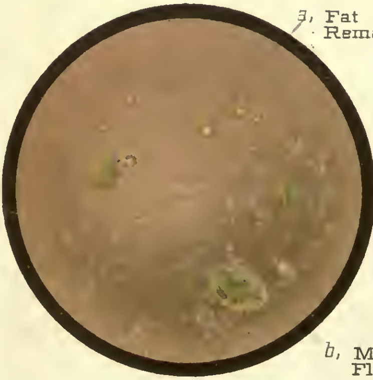

Fig. 5

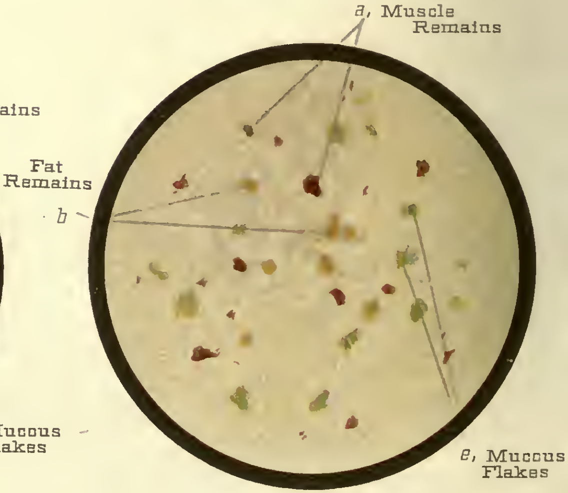

Fig. 6. Catarrh of the Small Intestine

THE EXAMINATION OF THE FUNCTION OF THE INTESTINES - - - 7

BY MEANS OF

THE TEST DIET

ITS

APPLICATION IN MEDICAL PRACTICE AND ITS DIAGNOSTIC AND THERAPEUTIC VALUE

BY

PROF. DR. ADOLF SCHMIDT
HALLE, A. S.

AUTHORIZED TRANSLATION FROM THE SECOND
REVISED AND ENLARGED GERMAN EDITION

BY

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PHILADELPHIA
F. A. DAVIS COMPANY, Publishers
1909

QY160
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1909

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[Registered at Stationers' Hall, London, Eng.]

Philadelphia, Pa., U. S. A.
Press of F. A. Davis Company,
1914-16 Cherry Street.

TRANSLATOR'S PREFACE TO NEW EDITION.

Since the publication of the first edition of Professor Schmidt's "Die Funktionsprüfung des Darmes mittelst der Probekost," this method for the examination of the function of the intestine by the test-diet has been widely adopted. While in this second edition some modifications of the original procedure are suggested, the main premise as to the value of the examination of the feces is maintained. Professor Schmidt has carefully reinvestigated this whole subject, in which he is admittedly a pioneer. He has added, in this edition, new material regarding intestinal disturbances due to a perversion of the function of the stomach, of the liver, and of the pancreas, which may be regarded as constituting a substantial contribution to the development of gastro-enterology.

Charles D. Aaron.

Detroit, Michigan.

PREFACE TO THE SECOND EDITION.

SINCE the publication of the first edition, the theoretical and practical results of the systematic examination of the feces by means of the test-diet have become so essentially extended, that a revision of most of the sections has become necessary. I seize this opportunity to add a number of illustrations which, I hope, will further facilitate the introduction of my method. To attain the same end the test-diet has been simplified by the introduction of a general plan. Numerous communications from colleagues and also the wide popularity that the "Functional Examination" has gained in other countries (translation into Russian by *Breitmann,* St. Petersburg; into English by Dr. *Aaron,* Detroit) show me to my satisfaction that my suggestions are practical.

ADOLF SCHMIDT.

Halle a. S.

PREFACE TO THE FIRST EDITION.

The following pages owe their origin to a series of lectures, which I delivered in the summer of 1903 at the instance of my colleagues. They sum up for medical purposes the results of my investigations and observations continued for about eight years. The desire to obtain an examination for the function of the intestines, *that could be carried out in practice,* analogous to the usual examinations of the stomach contents, and to advance by means of this examination the diagnosis and therapeutics of the diffuse intestinal diseases, in the same way as it has already been accomplished in the pathology of the stomach, was the incentive which led me from the beginning. The great deviations from our plan, which my investigations and those of my co-worker, Dr. *Strasburger,* made necessary in order to secure a scientifically established basis for our practical methods, have made it impossible to put this goal in the foreground, as I had it in mind to do. For this reason and partly also because our works were scattered in the inaccessible literature, our methods for examining the function of the intestine have not yet been able to secure a firm footing in practice. To many they at first seemed too complicated, a fault, which, I hope, has been almost altogether removed. The conviction, that our functional examination, though it still has many gaps, can be of service to the practitioner in many respects even in its present form, and the encouraging approbation of colleagues, have induced me to make this comprehensive presentation. At the same time it affords me a desired opportunity for the publication of some recently obtained results.

ADOLPH SCHMIDT.

Dresden.

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I. THE EXAMINATION OF THE FUNCTION OF THE INTESTINE.

Functional Diagnosis of the Stomach and of the Intestine.—Experiments in Digestion.—Conditions of a Rational Test of the Function of the Intestine.—The Examination of the Content of the Stomach as Indicator.

To any one who compares the present state of intestinal pathology with that of the pathology of the stomach, there can be no doubt, that in spite of the progress we have made, especially in the field of local inflammations (appendicitis), of stricture and of obstruction of the intestine, the former has nevertheless remained very far behind the latter. This is especially true of those diffuse diseases of the intestine and its adnexa (the liver and the pancreas) which have no readily recognizable symptoms,—and will form the subject of our consideration primarily—in other words, of the lighter grades of inflammation and the numerous other so-called functional diseases, for which it has hitherto proved impossible to find an anatomic condition. In the pathology of the stomach it is in the knowledge of just *these* forms of diseases that extraordinary progress has been made in the last two decades, and this progress we owe to the use of the stomach-tube for the purposes of diagnosis *(v. Leube)* and to the introduction of the test-breakfast and the test-meal *(Ewald, Riegel),* in short, to the creation of a functional diagnosis of the diseases of the stomach.

The aim of *functional diagnosis,* which is now being striven for in all branches of internal medicine, is the recognition of disturbances in the physiologic process (the functions) of organs, even when other objective signs of the disturbances are still wanting, when perhaps only indefinite subjective sensations are present or even no symptoms at all, as happens, for example, in *achylia gastrica.* In order to obtain this, an arbitrarily chosen task is imposed on the particular organ and from the way it discharges this task, its efficiency is inferred. The difficulties in functional diagnosis, which we encounter owing to the natural limitation of our anatomic knowledge and of our sense-perceptions, lie in finding suitable criteria that can be introduced into general practice, for testing the total work, or the functions of the separate organs and the demarcation of the ranges of physiologic fluctuation of these operations. These must naturally be accurately known before we can infer any pathologic conditions. Such a criterion once obtained, we could by means of it advance our diagnostic and therefore our therapeutic knowledge considerably. This is proved by the functional examination of the eye by means of the altogether arbitrary concept of visual acuity; this is proved for internal medicine by the advances in the pathology of the stomach. As a result of investigations with the stomach-tube, we are now in a position to control the work of the stomach in its finest details, and to test separately the part played in it by its three chief functions, motility, secretion, and absorption.

In intestinal pathology we are still far from this ideal. Owing to the inaccessibility of this organ we must forego the introduction of tubes and similar instruments to aid in palpation and illumination of the mucous membrane and the removal of chyme for test-purposes. Such methods can be employed only from the anus and may give us our bearings as to the condition of the rectum, but much more rarely as to the upper sections of the colon. *Hemmeter*

(1) and *Kuhn* (2) have repeatedly passed the pylorus by way of the stomach, but a general application of their methods is out of the question. Similarly the obtaining of duodenal secretion from the empty stomach, to which *Boas* (3) first called attention, and which *Volhard* (4) has recently attempted to simplify by the "oil-test-breakfast," has little prospect of being introduced into practice. Aside from the aids of physical diagnosis, of inspection, palpation, percussion, and transillumination of the abdomen, and from the evidences which are furnished by the examination of the urine and of the other organs in general, we are, in the main, dependent upon the examination of the feces.

But the examination of the feces is used entirely too seldom and valued too little. I believe I offend no one by asserting that, aside from hospital physicians and specialists on the diseases of the stomach and intestines, scarcely any practitioner proceeds in the investigation of the feces differently from a layman: *i.e.,* he inspects it and smells at it, and, at most, stirs it with a stick. Why is this? Is the prudery of the public and the repulsiveness of the examination of the feces the cause, or does the analysis of the feces in reality promise such slight results as not to be worth the trouble? I believe that both points enter here; for since *Nothnagel's* investigations, dating from 1884 (5)—the valuable results of which have unfortunately found admission only to a limited circle of physicians—to the very present time, the clinical analysis of feces has made scarcely any progress, and it cannot be denied that the method of examining the feces used at present only rarely gives results useful for diagnosis.

Still we must not forget the great advances which we owe to the numerous *experiments in metabolism* or *digestion* performed on normal and pathologic intestines. The experiment in digestion is the given criterion for the examination of the function of the intestine and its adnexa; it shows us how much and what constituents of the food leave the body undigested. We owe to the experiment in digestion practically everything that we know of the functional disturbances of the liver and the pancreas with regard to their secretion. It has also proved itself valuable for the recognition and differentiation of various intestinal disturbances; but (and that is the difficulty) it requires too complicated an apparatus to be adopted by all physicians; its application will always be confined to clinics and large hospitals.

Experiments in digestion have also other defects, which are not to be passed over in silence, when we are concerned with the possibilities of a functional test suitable for clinics (6). In the first place, the choice and composition of the nutriment is of decided importance for the amount of the food-remains that appear in the feces. In previous experiments, however, the choice of diet was in many respects made without regard to the efficiency of the intestine; at any rate there was never any question of uniformity in the diet—an absolute requirement for comparative investigations. Then through the usual chemical methods for determining the nitrogen and fat in the feces, the remains of albumin and fat from the food which go to waste were not indicated with sufficient accuracy. They were always measured together with the substances containing nitrogen and the fat-bodies excreted from the body itself, and we had no means of separating these two altogether heterogeneous groups from each other. Now under certain circumstances, especially in the case of a diet that contains little waste material, the excretions from the body constitute a very considerable portion of the content of nitrogen and fat in the feces. In a mixed diet, *e.g.*, the nitrogen that comes from the body amounts, according to *Rieder,* to about 29 per cent. of the total content of nitrogen in the feces (7), and *Strasburger* (8) has calculated, that with an easily digestible diet at least half of the entire nitrogen in the feces is to be credited to the fecal bacteria. As regards fat, *Rubner* (9) found 3 to 6 grams of ethereal extract in the daily feces, even with an almost fat-free diet. These figures prove that the results of the chemic analysis of the feces are quite unreliable, and it is obvious that it will fail altogether where we are concerned with the investigation of slight deviations from the normal or the finer disturbances of intestinal activity. To this is to be added the fact that till very recently we did not have any tolerably useful method for determining the carbohydrate remains in the feces. The method developed by *Strasburger* (10) in 1901 first proved sufficiently reliable and sensitive, but it is so difficult, as to demand the hand of an experienced chemist.

A third radical defect of the chemical analysis of the feces consists in the fact that it does not give us any information as to the form in which the undigested food-remains leave the body. A question that constantly forces itself upon us in the functional examination of the intestines is this: Are the undigested remains which are evacuated with the feces absolutely indigestible waste matter, or have nutritive elements, which ordinarily should have been digested, been evacuated at the same time. For the clinician it is not a matter of indifference whether the albumin-remains in the feces come from undigested muscle fibers or from connective tissue (tendinous fibers), and whether the undigested starch granules are free in the feces or are protected from the influence of the digestive juices by cellulose walls.

Concerning these matters only the macroscopic and microscopic investigation can give us information, and it would certainly be absurd to abandon these simple methods for the sake of chemic analysis.

The question suggests itself whether, retaining the principle, in itself correct, of the experiments in digestion, but with a simplified technic, and avoiding the sources of error here enumerated, it would not be possible to find a test of the function of the intestine that would be available for general practice. If this thought is followed out further, we have at once these points:—

1. *We must have as our starting point a uniform composition of the feces—a normal feces.* This normal feces need not, however, have so wide a scope as *Praussnitz* (11) would have it, as if it could come from any food at all, as long as that contained little waste matter, but it must always presuppose *one and the same uniformly constituted food, the "test-diet."* Everything depends upon the proper selection and general applicability of the test-diet. Any one who has ever made investigations of feces knows that, so far as the amount and kind of the remains are concerned, the significant conditions found depend primarily on the amount and kind of food that has been served, and only secondarily on the conditions of the digestive organs. At least, I would regard it as fallacious, under all circumstances, to draw inferences with regard to the functioning of the digestive organs from the condition of the remains of food substances in the feces, without first knowing exactly what food has been taken in the last two days. Just in the changing composition of the diet lies the difficulty of making the results of the investigation of the feces of value for medical practice. One need only examine his own stool macroscopically and microscopically regularly, for a few

days, especially after dinner, or supper, and he will wonder at the variety of things that pass the intestinal canal undigested. To those interested in the variable forms of human excrement, the excellent book of *van Ledden-Hulsebosch* (12) gives the desired information.

2. *The investigation of the stool must be simplified as much as possible.* All methods which cannot be carried out in practice are more or less worthless. They may be of great service for scientific investigation, but they will never be used among physicians. We need only to study the examination of the content of the stomach and see what few tests we can depend upon. Of all the numerous methods for determining acidity, only the phloroglucin test, the lactic acid test, and the total acidity test are used. Every other test has been abandoned in the clinics. The same thing is true as to the examination of the urine. Of the innumerable chemical tests only the simplest albumin and sugar tests have become the common property of physicians. Similarly in microscopy, only the making of the simplest preparations and stains comes into consideration. It may appear strange that I should lay so much stress on simplicity in the methods of examining the feces, since the "fermentation test" and the "digestion test," which were first proposed by me, have been criticised as being too elaborate. But as I have already pointed out in the preface, I have in the course of time learned to simplify my methods of investigation more and more. I am willing to admit that those tests in their original form were far too complicated, but I may urge in extenuation that only by the roundabout ways of the most exact detailed investigation can results be obtained for general utility. Now, when we know clearly the extent and fluctuations of the hydrochloric acid secretion of the stomach, having applied the most dif-

ficult chemic methods one after the other, we are able to make proper inferences as to the secretory capacity of the stomach from the simplest color-reactions. Such, too, will be the course in the investigation of the feces.

The examination of the stomach-content cannot be too closely kept in sight as an indicator. That method of functional examination of the intestine will be best which attaches itself as closely as possible to the functional examination of the stomach and which can ultimately be combined with it; for it is indeed well known that not a few intestinal disturbances have their starting point in the stomach, and that it is under all circumstances necessary to have a definite knowledge of the work of the stomach before proceeding to the investigation of the function of the intestine. The examination of the functional capacity of the intestine must in the first place unfold a complete picture of the digestive activity; furthermore, it must inform us what groups of food-stuffs, in the given case, are, especially or separately, defectively digested. Naturally in connection with the examination for undigested food-remains, attention must also be given to the pathologic products of the intestinal wall, so far as they are recognizable in the stool (mucus, pus, blood, etc.), and to the intestinal parasites, for it is impossible to draw an absolute limit between the organic and functional affections of the intestines. Furthermore we must have in view as a goal to be sought, though it is still far distant, a separate examination of the intestinal activity according to its chief functions, motility, secretion, and absorption. Finally, we must seek to separate the disturbances of motility according to the main segments of the intestine (the large intestine and the small intestine), from the disturbances of the various secretions, that coöperate in intestinal digestion, stomach, liver, pancreas, and intestine.

The following presentation will show that thus far we have not yet advanced beyond the beginning of these problems of ours; they will, however, inspire us with the hope that we can make progress on the way we have entered. After the discussion of the test-diet and the methods of examining feces, we will turn to the diagnostic and therapeutic application of the functional examination in diseases of the digestive organs—the diffuse, inflammatory, and functional diseases of the intestine and its adnexa.

II. THE TEST-DIET.

The New General, and the Older Detailed Test-Diet.—Possibilities of Another Test-Diet.—Objections to our Test-Diet.—Irritative Effect of Milk on the Intestine.—Imitations and Mutilations of the Test-Diet.

The requirements which must be imposed on a suitable test-diet are very manifold; it must be so selected that it can be taken by healthy persons as well as by most of those suffering with intestinal disorders; it must be as free as possible, but not absolutely free, from waste matter, in order that the stimulus ordinarily furnished by the ingesta should not be completely absent; it must be capable of supplying the minimum, at least, of calories required by the body (at rest) and must contain the three chief groups of food-stuffs in proportionate relation to each other; finally, it must be of simple composition, easy to make and uniformly prepared. Only when all these general requirements are fulfilled, the narrower selection with regard to the methods of subsequent fecal examination can proceed.

The first prescriptions, as to the test-diet, which *Strassburger* and I suggested (6, 13) had later to undergo manifold modifications, in correspondence with the progressive methods of the examination of the feces. While at the beginning we laid the main stress on the "fermentation-test," there was added later on the "meat-test" (14) and others. Furthermore it became evident in the course of time that for the purposes of practice it is not altogether necessary to carry out strictly the quantitative measurement of each respective constituent, as we did for a long time for the purpose of establishing exactly its normal values. A knowledge of this latter condition particularly, which I was able to ascertain in most recent times by special experiments on patients suffering from dyspepsia (15), due to acidity, have enabled me to simplify more and more the prescription of the test-diet, *so that it can be used without difficulty even in ambulatory practice, in the form in which I now apply it.* Thus I hope to overcome entirely the difficulties which, according to the assertion of *Boas* (16), of *Strauss* (17), and of others who are experienced authorities in the pathology of digestion, have been in the way of the general introduction of my method until the present time. Still I wish to emphasize that in the cases where a clinical observation is desirable, or where it is necessary to carry out an exact, quantitative analysis of the feces, I apply still to-day the detailed prescription for the test-diet as described in the first edition of this book and I give it here again in connection with the more general prescriptions:—

General Test-Diet.

In the morning: ½ liter {milk or tea or cocoa} if possible with much milk,
together with 1 roll and butter and 1 soft-boiled egg.

Breakfast: 1 dish of oatmeal-gruel cooked in milk and strained (salt or sugar permissible); [gruel or porridge may, under certain conditions, also be given].

At noon: ¼ ℔ finely chopped lean beef, broiled rare, with butter (the interior raw),
along with it not too small a portion of potato-broth (well strained).

In the afternoon: same as in the morning, but no egg.

In the evening: ½ liter milk or 1 plate of soup (as in the morning),
together with a roll and butter and 1-2 soft-boiled eggs (or scrambled).

It is obvious that the scope of this prescription is very broad, especially if, as is permissible, the meals are occasionally interchanged or combined. Under certain circumstances, further concessions may be made, *e.g.*, some wine, some coffee added to the milk, or bouillon at noon, some chopped cold, roast veal in the evening. The following four fundamentals of the test-diet, however, must be strictly observed:—

1. A certain moderate measure of milk (½-1½ liters), which, however, may be boiled entirely with the foods;
2. about 100 Gm. *white bread* (or zwieback, cakes, etc.);
3. a good portion (100-250 Gm.) of *potato-broth;*
4. ¼ ℔ chopped *beef,* a *portion,* at least, of which must remain *raw* or *half-raw.*

If we compare this general prescription, which I prescribe for less serious and ambulatory patients, with the detailed test-diet described below and which I used exclusively for years, it will easily be seen that, though it disposes of most of the quantitative measurements which are not always practicable for the household, it does not appreciably deviate from its fundamentals.

Detailed Test-Diet.

(For Clinical Purposes and Quantitative Analyses).

In the morning: 0.5 liter milk [or if milk does not agree, 0.5 liter cocoa (prepared from 20 Gm. cocoa-powder, 10 Gm. sugar, 400 Gm. water, and 100 Gm. milk)],
with 50 Gm. zwieback.

In the forenoon: 0.5 liter oatmeal-gruel [prepared from 40 Gm. oatmeal, 10 Gm. butter, 200 Gm. milk, 300 Gm. water, 1 egg (strained), and some salt].

At noon: 125 Gm. chopped beef (raw weight), broiled rare with 20 Gm. of butter, so that the interior still remains raw,
to this 250 Gm. potato-broth (made of 190 Gm. mashed potatoes, 100 Gm. milk, and 10 Gm. butter and some salt).

In the afternoon: as in the morning.

In the evening: as in the forenoon.

Accordingly, this diet consists of 1.5 liter milk, 100 Gm. zwieback, 2 eggs, 50 Gm. butter, 125 Gm. beef, 190 Gm. potatoes, and gruel made of 80 Gm. oatmeal. It contains about 110 Gm. albumin, 105 Gm. fat, and 200 Gm. carbohydrates, or a total of 2247 calories (raw calories),[1] and thus it answers the minimum requirement of calories necessary for an adult at rest. *The test is generally given*

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[1] The figures here given are on the basis of *Vaughan Harley* and *Francis W. Goodbody's* (18) analyses and differ slightly from the figures given in the former edition of this book, which were computed after the tables of Koenig. We deal with merely assumed averages.

for three days, sometimes longer, at any rate until a stool is obtained, which comes with certainty from this diet. With a normal condition this occurs at the second defecation after the beginning of the test-diet. The patient, as a rule, does not need to take a special powder in order to mark off the normal excrement from the excrement of the former diet, for the normal excrement can generally be recognized at once by its uniform consistency and its lighter color. Still, where this is desired, or where the total excrement of three days is to be collected, I administer at the beginning and end of the test-diet a wafer with 0.3 Gm. powdered carmin, which passes the digestive canal unchanged and gives a distinguishing color to the corresponding part of the feces; only some very sensitive intestines are sometimes irritated by it.[1]

It is obvious that this test-diet does not represent the "only possible" or "only conceivable" form of diet for the functional examination of the intestine. To make such a claim never entered my mind. Within the above-described limits of the necessary conditions there are very many diet lists suitable for the clinical examination of the function of the intestine, and it may therefore again be expressly stated that we are here dealing with an altogether arbitrarily chosen standard. But what gives significance to just *this test-diet* is the fact that by means of it (or unessential modifications of it) all fecal investigations were carried out by *Strasburger* and myself; that it is the foundation of all the tests which will be described later;

---

[1] If diarrheas exist and the evacuation for a definite period is to be collected, the experiment must be based on a greater number of days, for the sharp demarcation of the red from the uncolored parts is not possible. Consequently an error creeps in, which can be overcome only by the extension of the test over a greater period of time.

that it has furnished the basis of numerous quantitative analyses and experiments in digestion, which are given in our "Pathology of the Feces" (23) and to a certain extent form the basis of the entire coprology. *It would, therefore, be foolish to dispense with this test-diet, or to desire to substitute another for it, without a cogent reason. Do such reasons present themselves?*

Of the two essential objections which were hitherto raised against the test-diet, one refers to the *difficulties in application,* as already mentioned. Either it was asserted that the preparation of the test-diet is too elaborate and, therefore, possible only in such a clinic as is equipped with a kitchen for that purpose, or the patients were not willing to content themselves for three days with such an insipid diet.

I will not deny that good-will on the part of the patient and a certain determination, if not persistence, on the part of the physician, are indispensable to the full application of the *detailed test-diet* (and only to this does the objection apply) ; nor that a good kitchen is a necessity for an even and tasteful preparation of food. But I have never encountered any real opposition, either in hospital or in private practice, although I have examined many hundreds of patients and also healthy persons, by means of the test-diet. One must first himself be convinced that in most cases no results can be attained without the test-diet, and then the patient and the cooks will be convinced of it. I can state to my greatest satisfaction that this conviction is now almost generally shared by the competent practitioners. I refer merely to remarks relevant to this matter made by *Boas* (16), *Strauss* (17), *Rosenheim* (19), *Kuttner* (20), *Selter* (21), *Gaultier* (22), *Harley* and *Goodbody* (18), and others.

Nevertheless, I admit that it is exceedingly desirable to widen the scope of application of the test-diet through further simplifications, so as to make it accessible to general and ambulatory practice. This is the reason why I have treated first of the *general test-diet,* which can be employed even under such difficult circumstances as those of poly-clinical practice. It affords also the possibility of making the diet more suitable to the taste and habits of the patients of various social classes and of various countries. In this respect the statement of *Harley* and *Goodbody,* that the English patients show a particular aversion to the lack of variety in my detailed description, was very instructive. The same applies to Americans, as I gather from personal communications with some colleagues. The French public, on the other hand, seems to be more easily satisfied with a flat diet (cf. *Gaultier*), and *Oerum* (24) reports from Sweden that my diet is usually "very readily" taken. In the new *general* prescription the variety of diet is so enlarged that even this difficulty appears to be removed.

The second objection is in regard to *the milk used in the test-diet.* It is claimed that milk is not fit for a normal diet, because many adults cannot tolerate it, since it causes diarrhea. This objection has also a certain justification, for it cannot be denied that, indeed a number of persons suffering from gastric and intestinal disorders, and even healthy persons, soon after the use of milk are affected by diarrhea, or—and this is especially noteworthy—by constipation.

Very remarkable in this connection is the fact that it is almost always pure milk (raw or boiled) that presents these characteristics, whereas the addition of milk to other foods (milk cooked along with foods) rarely produces such results. Indeed, many times merely a slight addition to the milk (cognac, cocoa, rice, barley water, starch) or even a different form of serving it (taking it in sups instead of drinking it, warming, boiling, etc.) suffices to prevent those disturbances. In most cases, moreover, the diarrhea or constipation represents only a temporary phenomenon, which stops of itself after 2 to 3 days. It is easily imaginable that in these cases, where the patient has not been accustomed, for a long time, to drink milk, the digestive canal and its flora require a certain amount of time to adapt themselves to the new diet. For the remaining cases we have as yet no satisfactory explanation of the peculiar effect of milk on peristalsis. The cause cannot lie in the ingredients of the milk itself, the mildest and least irritating foodstuff, which is tolerated by even the delicate digestive canal of the new-born babe. Perhaps in such persons it is decomposed more readily than usually and irritates the intestine through the increased products of decomposition, or the intestine, which is in itself already irritated, is more sensitive than the healthy intestine to the normally formed products of decomposition.

The question is, in the first place, whether the incidental effects of milk on the intestine are indeed so frequent, that we may not include it in the test-diet at all. My own experience on this point is to the effect that the doubts expressed are very much exaggerated, and that under no circumstances are we to follow *Rosenfeld* (25) who puts milk down summarily as a substance irritative to the intestine. Among our numerous experiments with the test-diet, we have had to give up the investigation on account of the irritative effect of milk only very exceptionally (perhaps in 5 to 8 per cent.). I disregard here the cases, which are not very rare, where the diarrhea or constipated effect extended only over the first 1 to 2 days, and I can only advise, that in case such disorders appear, the experiment should not at once be dropped, but prolonged at least for 4 to 5 days, before it is finally abandoned. The assertions of patients, that they cannot tolerate milk, etc., should not be given immediate credence; very often just such patients later tolerated milk very well. Where irritative effects really persist for a considerable time, it is very often, so far as I can judge, a case of tuberculosis of the intestine, a disorder which is especially characterized by a capricious alteration of constipation and diarrhea and by the fact that it is influenced by dietetic prescriptions only to a remarkably slight extent.

In the present *general form of the test-diet,* not only is the quantity of milk greatly reduced, but the larger part of it is boiled in the foods and the rest can be disposed of by discreet dosing of tea and cocoa. I have never seen any irritations result from the milk contained in the oatmeal-gruel or potato-broth, and the majority of those that cannot tolerate milk will tolerate milk-cocoa or milk-tea. In exceptional cases this may be left out; but in the usual treatment the milk cannot be omitted from the test-diet. In my opinion no suitable test-diet can be prepared at all without milk. For the amount of starch and of meat in my test-diet for the purposes of fecal investigations does not allow an increase, and under these circumstances how are we to produce the needed amount of calories by means of sugar, eggs, or other artificial nutritive preparations, without coming upon new difficulties? Turn the matter as we may, a uniform test-diet that would be suitable for most cases, seems to me impossible without some milk as an ingredient. Besides (and this is a second important point), milk is and remains that kind of food-stuff which, while possessing sufficient nutritive value, requires relatively least chemical and mechanical digestive labor. It is the food-stuff to which the disturbed intestine must therefore accustom itself again, if this is at all possible, in order to return to a healthy state. For me it is the first therapeutic principle in all catarrhal and functional disturbances of intestinal activity to strive to make milk or milk preparations (buttermilk, kephyr) tolerable again, and I regard the test-diet therefore not only as a diagnostic, but, at the same time also, as a therapeutic aid, which in many slighter affections suffices by itself to bring about a cure. *Strasburger* and I (26) pointed this out, when we described fermentative dyspepsia, and my further experience is to the same effect.

Before I close this chapter, I wish to mention briefly those authors that attempt to "simplify" or to "improve" my test-diet, or to modify it for their special purposes. Thus *Zweig* (27) and *Strauss* (28) eliminate my meat-prescription from the test-diet, and order as a special "meat-test" 100 Gm. chopped raw meat to any kind of diet. I consider such a mutilation of the test-diet utterly false. Besides, I prefer to have the meat broiled in some butter over a strong fire, because many have an aversion to the taste of raw meat. Likewise the "bead-test" of *Einhorn* (29), though it is proclaimed as a new method, is a mutilation and not a simplification of my test-diet. It consists of a number of test-substances (catgut (!), fish-bones (!), meat, potatoes, mutton-fat, thymus) tied in small portions to a string of glass-beads, and is given with the usual diet in a gelatine-capsule. After the passage, the string of beads is extracted from the feces and examined to ascertain to what extent the separate substances have been digested. It is clear that the principle of this procedure is taken from my functional test. I object to this technic, because it does not take the physiologic relations into sufficient account. The point in question is not whether a small portion of meat or potatoes can be converted by the digestive juices, but whether the digestive organs are equal to their normal efficiency. The former may still happen, when the latter no longer occurs. Therefore the entire diet must be prepared in accordance with the demands of the test, and the entire respective feces must be examined. Besides it is known that in sensitive intestines even a few beads are sufficient to increase the peristalsis considerably.

Vaughan-Harley and *Francis W. Goodbody* (18) have gone into the question of the test-diet very thoroughly. I have already mentioned that they have found my test-diet, despite its acknowledged advantages, too monotonous for the English palate for any length of time. They composed therefore a "mixed diet" of their own, and I wish to remark with regard to it, that it omits the chopped raw meat and potato-broth, the two nutriments which are the most important for the functional test. It is, therefore, not fit for the functional test to be made by means of the simple technic of the examination of the feces which I have worked out (see next section). It may, however, be used, like any other flat diet, for tests as to quantitative digestion.

R. Gaultier (22), in France, turned his attention to the analysis of the feces and to the test-diet. His book is not more than a distorted reconstruction in French of the coprology by *Strassburger* and myself, as I have already pointed out in a review of it in the "Zentralblat für innere Medizin" (30). His own, and, as he claims, the real method of the functional test, is nothing else but a somewhat modified and perverted imitation of my test. I prefer, therefore, not to discuss *Gaultier's* test-diet any further.

Finally, I would mention here the desmoid-test of *Sahli* which, according to the author's own statement, is based on the test of the connective tissue as set forth by me, and which is meant to replace the latter when the stomach is involved in the digestive disturbance. I shall revert to this more fully in the chapter dealing with the gastrogenic disturbances of the intestines.

III. THE EXAMINATION OF THE FECES.

As soon as the evacuation, after the beginning of the test-diet, has taken on the easily recognizable characteristics of the "normal excrement" (uniform brown color, soft but firm consistency) (s. Fig. 12, Plate III), which generally occurs in the second or third defecation, we proceed with the investigation. If the time of passage of the ingesta is to be ascertained, which I do not consider as important as other authors do, carmin must be given (see p. 14). I do not regard it superfluous to remark here that when patients cannot deposit their excrement in suitable transportable vessels (commodes, etc.), there should be placed at their disposal glass jars about 15 centimeters high and 8 centimeters in diameter, with screw-top glass covers, or fruit-jars and a number of flat wooden spatulas in order to spare them the discomforts of the unnecessary manipulations. *These glass jars should be sent for examination immediately after evacuation. This investigation should be carried out on feces as fresh as possible.* If carried out in the manner subsequently to be described, it requires at most ten minutes and only very simple apparatus. In all respects, therefore, it may be regarded as similar to the ordinary examination of the urine and of the stomach-content.

A. Macroscopic Examination.

The macroscopic examination forms *the most important part of the whole procedure,* and that alone is often sufficient to enable the experienced observer to form a judgment. In the first place it determines whether *color, consistency,* and *odor* correspond with the normal feces. As to *color* it is to be remarked that, if milk is taken with the test-diet, it will be light-brown, whereas when cocoa is taken it will be red-brown. Of deviations through disease only tar-feces (blood) and clay-colored stool (fat) come under consideration. The latter especially cannot fail to be recognized, since its coherency (clay) is characteristic. As to the odor which, under ordinary conditions, should be mildly excremental, let it be remarked briefly that under pathologic conditions it may become butyric acid-like, or even biting-sour (acetic acid), or it may become malodorous through putrefaction. Furthermore, there are recognizable, easily at the first inspection, *gross flakes of mucus, blood-pus, portions of tapeworm, and other parasites, stones, remains of eggshells, and other foreign bodies.* It is not necessary to discuss these here any further.[1] Only this much is to be remarked, that it is of importance whether the mass of the feces itself is thoroughly mixed with mucus, pus, or blood, so that both parts cannot be easily distinguished, or whether the mass is not thoroughly mixed so that both parts may be easily recognized. The latter point is decisive for a low origin of the admixtures (rectum, flexure).

We proceed after this to a *closer examination.* For this purpose the entire excrement is first stirred up thoroughly with a wooden spatula, and a small amount of about the size of a walnut is transferred to a porcelain mortar (about 12 centimeters in diameter). In this it is ground up as fine as possible with the addition of, at first, a few cubic centimeters and gradually more distilled water (to about the consistency of sauce). The grinding up must be done carefully, so that no coherent particles of excrement remain. The ground-up specimen of excrement is then spread over a flat black plate or a large vessel in as thin a layer as possible and account is taken of all the elements that are still recognizable in it by the naked eye.

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[1] Concerning the evidence of their presence and of their semiotic significance compare "Die Fäzes des Menschen," 2d ed., p. 35 ff.

This method of macroscopic examination (through grinding) is, for the purpose of the functional test, the only rational one. It deserves preference, by far, to the laborious cleansing [*van Ledden-Hülsebosch* (12), *Strauss* (31)] or washing through sieves [*Boas* (32) and others]. The latter is of use only when parasites, gall-stones, or foreign bodies are being directly sought for.

The normal test-diet stool, after having been ground up very small (smaller than pinheads) shows brown or red-brown points (chaffy remains of the oatmeal-gruel, remains of cocoa-nibs), the nature of which is ultimately explained by a microscopic examination. Otherwise it is entirely homogenous (see Fig. 1, Plate II).

[If, after grinding-up, some remains of vegetables or fruit are found, we have still no evidence that the patient did not observe the test-diet strictly. They may have been taken days previous to the test; for it is a well-known fact that the last remains of a former diet may still appear under examination in the feces even several days afterwards.]

Under pathologic conditions there appear:

Food-remains:

1. *Remains of connective tissue and tendons from the chopped meat eaten* (see Fig. 2). These are frequently found and can at once be distinguished from the mucus by

their whitish yellow color, their thread-like appearance, and their solid consistency. In doubtful cases a thread is placed under the microscope and treated with acetic acid; in connective tissue the thread-like structure disappears, whereas in mucus it appears then and then only. *Isolated little tendon fibers are occasionally found even with normal digestion. As soon as they occur more numerous and larger, a disturbance of digestion, namely of gastric digestion, is involved.*

2. *Remains of muscle tissue* (see Fig. 3 and Fig. 6a). Chiefly very small, brown colored rods, looking like splinters of wood; can be broken up by pressure and show muscular structure under the microscope. *The muscle-remains point to a disturbance of intestinal digestion.*

3. Occasionally remains of connective tissue and of muscle occur combined as meat-remains.

4. *Potato-remains.* The potato-remains appear like sago-grain, glassy, transparent granules, and are easily mistaken for flakes of mucus[1] (see Fig. 4). They are readily discovered by their globular appearance and larger consistency of the fecal mass. In rare cases, and almost the thinly spread layer of excrement, whereas the mucus spreads out flat. Crushed they show, under the microscope, the potato cells with remains of starch granules colored blue with iodine.

5. *Fat-remains* (see Fig. 5a and Fig. 6b). Their presence is evidenced by the light color and by the clay-like consistency which causes them to project above the level of always only in cases of severe diarrhea, there appear also small, light-yellow, and soft lumps.

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[1] Former and even late investigators erroneously described these as mucus (23, 33).

Accidental constituents (only the most important need be noted).

6. *Mucus, in larger and smaller, even very small flakes* (see Fig. 5b and Fig. 6c). In the process of grinding up the excrement the mucus, when glassy and soft, is slightly broken up, but not dissolved. Larger shreds, or strips, tubes, moniliform structures, etc., as in the case of enteritis membranacea and colica mucosa, can be removed before the grinding up, so that only those substances that are contained in the excrement itself come to light. The recognition of large mucous flakes will scarcely ever occasion difficulties. Nevertheless, we should not forget that, if the mucus of the large intestine is thickly set with cells and fat-bodies, it may lose its original glassy character and soft consistency altogether and assume a white or brown color, with a gummy or even a leathery hardness. The smaller the flakes of mucus, the more difficult is it to recognize them, although, according to my experience, there is *no form of mucus found mingled with the feces, which on thorough inspection cannot be recognized even with the naked eye—especially if some of the ground-up mass is placed on a glass plate and held up against the light*—as glassy, transparent flakes, occasionally colored yellow by bilirubin, with irregular, ragged outlines. The mucous particles intermixed with the feces, formerly described by *Nothnagel* (5) and recognizable only by microscopic examination, the so-called "hyaline mucus-islands" and the yellow "mucus-granules" do not consist of mucus, as I have proved (23, 34). If any one is in doubt whether any little flake is really mucus, or wishes to make sure of the more minute structure of the mucous flakes (amount, kind of cells, parasite eggs, amebæ, crystals of bilirubin and Charcot-Leyden crystals, bacteria, etc.), which is often

desirable, he can place a little flake under the microscope and examine it—best without staining—with low and later with the *highest* power.

7. *Large crystals of ammonio-magnesium phosphate.* These are present in rare cases, in putrefying, mal-odorous stools; they grate when the excrement is ground up in the mortar, and can easily be recognized by their well-known coffin-lid shape and chemical reaction (solution in all acids).

[Stone cells of pears which may have been left over from a former diet, betray a similar conduct, namely they grate when the feces are ground up.]

B. Microscopic Examination.

The microscopic examination serves for the most part to complete the macroscopic; by means of it alone pathologic conditions are seldom disclosed. At all events it is well to carry it out in the following way: If the parts that are recognizable with the naked eye are eventually to be examined with the aid of the microscope, three preparations are made of the feces that was originally well stirred up—if it is too hard, a little water is added—*i.e.*, small particles are placed respectively in the middle and on either end of a slide. Without adding anything to it, the *first* is crushed into a very fine layer with the cover-glass. The *second* is thoroughly stirred with a small drop, 36 per cent. (official) acetic acid, and heated for a moment over the flame (until it begins to boil), and then covered with the cover-glass. The *third* is rubbed up well with a small drop of a strong solution of iodo-potassium iodide (iodine, 1.0; potassium iodide, 2.0; aq. dest., 50.0).

The normal excrement examined in this manner gives the following result: *In the first preparation* without any addition (cf. Fig. 7, Plate II) there is seen a detritus consisting of the smallest granules, globules, bacteria, etc., the more minute details of which cannot be recognized in ordinary inspection with strong dry lenses (Fig. 7*f*). The larger bodies imbedded in these are:—

Isolated fragments of muscle-fibers, yellow in color, *with rounded edges,* flake-like structures, occasionally with indications of cross striations (Fig. 7*a*); further

scattered smaller and larger yellow salts of calcium, flakes consisting of fatty acid salts of calcium with irregular borders, bright and deep yellow in color (Fig. 7*b*), and

colorless soaps (Fig. 7*c*);

isolated potato cells, either empty or with remains of a content, which can no longer be differentiated (Fig. 7*e*);

scanty chaffy remains from the oatmeal-gruel, often recognizable with the naked eye as little dark flakes (Fig. 7*d*);

finally, according to circumstances (where cocoa is taken instead of milk), *remains of cocoa* (Fig. 7*g*).

In the second preparation which serves to melt all the fat-remains temporarily and thus to furnish a view of the entire fat-content, there are seen, after cooling, *small flakes of fatty acid* scattered over the entire preparation. The larger salts of calcium and soap flakes have disappeared. If the preparation is again carefully heated over the flame and placed under the microscope while still hot, the small flakes of fatty acid are melted into drops, which congeal very suddenly, as the preparation cools again.

In the third preparation, which appears brown in color, the potato-remains ultimately assume a slight *violet* shade (not blue); the blue colored parts (starch granules or microbes) are absent.

The following conditions are pathologic (cf. Fig. 8):—

1. *In the native preparations:*

Fragments of muscle in larger number and better state of preservation (Fig. 8*a*).

These appear as longer bundles with cross striations *and sharp edges,* or as a collection of irregularly outlined homogeneous flakes (yellow granules).[1]

Needles of fatty acid and soap. Numerous little crystals permeating the mass (Fig. 8*b*).

Drops of neutral fat (Fig. 8*c*).

Numerous groups of potato cells with more or less well-preserved starch granules.

Parasite eggs (mucus, connective tissue, pus, and other macroscopic parts, of course, also appear).

2. *In the acetic-acid preparation:*

massive fatty acid flakes, in comparison with which the remaining components form a very slight part.

3. *In the iodine preparation:*

blue colored remains of starch granules (in potato cells or free) (Fig. 8*d*):

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[1] The *digestion test* of the feces previously described by me, which depended on a measurement of the quantity of undigested (macroscopic and microscopic) muscle remains, I abandoned later as too detailed and superfluous. The appearance macroscopically of muscle remains suffices decisively to prove a disturbance of intestinal digestion. *The microscopic* finding of many fragments of muscle fibers serves to supplement it; only in rare cases can the diagnosis be based on it alone.

blue or violet fungus spores, rods, or thread-like bacilli (Fig. 8);

yeast cells (which are colored yellow by iodine) (see Fig. 8*f*), sarcinæ and other striking micro-organisms (*e.g.*, lactic acid bacilli).

C. The Chemic Examination.

Of chemical tests only the following need to be considered for practice:—

The testing of the reaction, the sublimate-test, the incubator-test; furthermore, the blood-test and the examination for dissolved albumin. While the first three tests must always be made, the blood- and albumin-test are needed only under special conditions.

1. *For the reaction-test* a strip of red and blue litmus paper is placed on (not into) the excrement, which has been ground up with distilled water (for the exact macroscopic examination). Normally the reaction is amphoteric or *at most feebly* acid or *feebly* alkaline.

2. The *sublimate-test* (23, 35, 36) consists in placing some of the same feces, that has been ground up with water, in a flat glass vessel filled with a concentrated watery solution of $HgCl_2$, stirring it and leaving it stand (until the next day). The normal feces are colored red (hydrobilirubin), the more intensively colored, the fresher and less decomposed the excrement is. Particles colored green, even if microscopically small, are pathologic. They show the presence of unchanged bile pigment (bilirubin). The entire fecal mass, too, may turn green.

3. *The incubator-test,* as I perform it now, has been very essentially simplified as compared with the original

fermentation test. From the fresh excrement (which has been stirred up, but not yet thinned with water) a small amount[1] about the size of a walnut is taken with one of the wooden spatulas and put into the lower vessel of *Strasburger's* fermentation tube (Fig. 9*a*).[2] (In hard stools less is taken; in soft more; in fluid stools the lower vessel is filled entirely.) In this it is stirred up with water with the aid of a wooden spatula, and then the rubber stopper is put on, care being taken to exclude air bubbles. The rubber stopper is now taken from the little tube *b* and the tube filled with tap-water. This is then closed by turning over the vessel *a* together with the small stoppers, and the vessel *c* (empty) connected with it; *c* has an opening on top and acts as an ascension-pipe *(Steigrohr)*. Note is taken of the figure indicating how far the connecting glass tubes enter into the lumen of the three vessels. When the apparatus is put together, it is placed for twenty-four hours in an incubator heated to 37° C. If gas is developed from the feces within this time, this collects in *a* or *b* and a corresponding amount of water is driven into the ascension-pipe *c*. The height of the water in the ascension-pipe is noted, the vessel *a* is opened, and the reaction of it is tested with litmus paper, and compared with the reaction before the test was instituted. There exists a modification of the fermentation tube by *Dr. Baurmeister* (Braunschweig) which has the advantage of being handier and stronger (see Fig. 9*b*).

Normally no gas, or little, is formed in the incubator-test and the original reaction of the feces undergoes no

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[1] It should be about 5 Gm.; the estimated amount, however, is sufficient for our purposes.

[2] Can be obtained through *C. Gerhardt,* Utensilienlager, Bonn, Bornheimerstr.

important change (see Fig. 10*a*). *If gas is developed to such an amount that one-half or more of the ascension-pipe is filled with water, then pathologic conditions are involved, and if at the same time the reaction has become distinctly more acid, carbohydrate fermentation (the positive result of the fermentation-test)* (see Fig. 10*b*), *has occurred; if it has become distinctly more alkaline, albumin putrefaction has occurred* (see Fig. 10*c*).

In the former case the opened glass *a* gives off the odor of butyric acid, in the latter case it gives off a very decided putrefactive odor. The color of fermenting feces is generally brighter, that of putrefying feces is generally darker. *The suspicion of pathologic fecal fermentation can often be confirmed by the mere inspection of the original feces, namely, if they appear light brown and foamy, react acid, and smell of butyric acid.*

In rare cases the fermenting and the putrefying processes so interact upon each other that, notwithstanding the copious formation of gas, the reaction and the other characteristics (color, odor) will not change considerably.

4. *The examination for dissolved albumin* comes into consideration only in cases of diarrhea, and then only when it is questionable whether the fundamental disturbance is purely functional or organic (catarrh, ulcers) (see Chapter VII B). This examination requires somewhat more time and is carried out, according to *Schlössmann* (37), in the following manner, which is best and certain:

Out of the remainder of the fecal mass 20-30 Gm. are ground up with water which should be added gradually in small quantities, until thinned into a fluid consistency. Let it stand thus for several hours, and then filter through a double filter. The muddy filtrate is clarified by a filter prepared with some pure diatomaceous earth.

To this clarified filtrate there is added into the test-tube 36 per cent. acetic acid carefully, *in drops,* as long as a turbidity or a precipitate is formed. *This precipitate is the (normal) nucleo-proteid of the feces.* This is filtered best by a filter made of diatomaceous earth, and the new filtrate is *tested* with a drop of ferrocyanide of potassium *to ascertain the presence of dissolved albumin (and albumoses).*

5. *The blood-test* differs from the functional test in as much as its use is of value only in case of organic diseases, and *then it has for its condition a diet without meat.* If the blood-test is to be combined with the test-diet, it is necessary to add one or two days free from meat, after the stool of the test-diet has been deposited, and to examine the next stool as soon as possible.

For the evidence of blood I use exclusively the test by *Weber,* in the following manner:

About 5-10 Gm. feces are stirred in a mortar with water until it is of a thick, fluid-like consistency. (If the feces contain much fat, they should be covered with about three times as much ether and stirred thoroughly once more with the pestle. The ether should then be poured off.) To this thick, fluid-like excrement there should be added about a third as much of strong acetic acid, and the whole stirred up again. About 10 cubic centimeters of the entire mass are poured into a test-tube, and about the same quantity of ether is added to it, and is carefully mixed a number of times (no shaking). If, in spite of this, the ether does not settle well enough, one or two drops of alcohol should be added. (If much blood is present, the ether will show a red-brown color, and through the spectroscope the characteristic absorption bands of the acid hematin can be recognized.)

The ether is poured off and a few granulated grains

Fig. 7. Normal Test-Diet Feces (Combined Picture). (Leitz, Obj. 7.)

a, Muscle-remains.
b, Yellow calcium salts.
c, Soaps.
d, Chaffy remains.
e, Empty potato cells.
f, Detritus.
g, Cocoa remains.

Fig. 8. Pathologic Ingredients in the Test-Diet Feces (Combined Picture). (Leitz, Obj. 7.)

a, Large muscle fragments.
b, Fatty acids and soap needles.
c, Neutral fat.
d, Starch granules in potato cells.
e, Granuliferous clostridia and hyphomycetes.
f, Yeast cells.

Fig. 9 A. Fermentation Tubes. (Dr. Strasburger.)

Fig. 9 B. Fermentation Tubes. (Dr. Baurmeister.)

To this clarified [illegible] into the test-tube 30 per cent. acetic acid [illegible], as long as a turbidity or a precipitate [illegible]. This precipitate is the (normal) nucleo-proteid of the feces. [illegible] by a filter made of diatomaceous [illegible] is tested with a drop of ferrocyanide of potassium to ascertain the presence of dissolved albumin (and albumoses).

5. The blood-test differs from the functional test in as much as [illegible] diseases, and then it has for its condition a diet without meat. If the blood-test is to be combined with the test-diet, it is necessary to add one or two days free from meat, after the [illegible] of the test-diet has been deposited, [illegible] the [illegible] as possible.

[illegible] the test [illegible]

[illegible] with water until [illegible]. If the feces contain much fat, they [illegible] about three times as much ether and stirred thoroughly once more with the pestle. The ether should then be poured off.) To this thick [illegible] should be added about a third as much of strong acetic acid, and the whole stirred up again. [illegible] cubic centimeters of the entire mass [illegible] of ether [illegible] a number [illegible] does not [illegible] alcohol should be added. ([illegible] the ether will show [illegible] the spectroscope the characteristic [illegible] of the acid hematin can be recognized.)

The ether is poured off and a few granulated grains

Fig. 7 (Leitz, Obj. 7.) Plate II

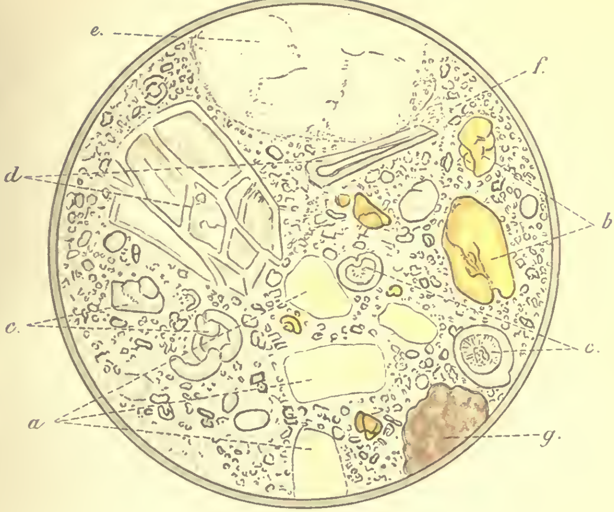
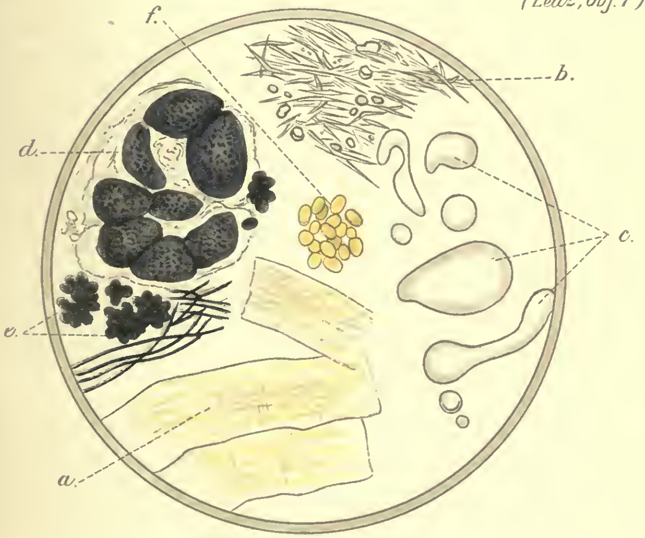


Fig. 8 (Leitz, Obj. 7)



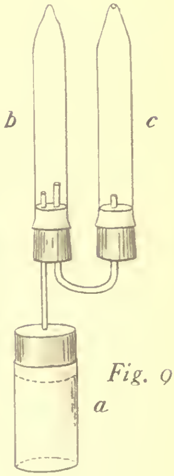


Fig. 9 A

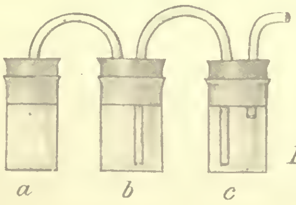


Fig. 9 B

of guaiac resin and 20-30 drops of 30 per cent. peroxide of hydrogen is added. The whole is shaken. A blue coloring shows the presence of blood, provided that there is no pus mixed with the feces.

D. The Bacteriologic Examination of the Feces.

The bacteriological examination of the feces does not fall within the scope of the functional test, and therefore it will not be discussed here further. If we disregard the causes of specific infectious diseases, as the cholera vibrio, and the bacilli of typhoid fever, tuberculosis and dysentery, the pathogenic ameba, and protozoa, there are occasionally found in intestinal diseases certain microbes which, abounding excessively in the feces, may be considered as the cause (proteus, pyocyaneus, Escherich's blue bacilli, streptococci, yeast, sarcinæ, etc.), *but in general the rule holds good in regard to the flora of the feces that it depends qualitatively as well as quantitatively on the constitution of the culture-basis.* In the cases referred to the condition is to be understood, that at first the pathologic mixture of the feces afforded the respective microbes an opportunity for development—these in some isolated cases sometimes pass through the intestine—until their permanent retention led to inflammatory processes and thereby to an independent disease sustained by the abundant multiplication of the microbes.

In the normal test-diet stool the coli bacilli preponderate so greatly that alongside of them other kinds cannot come into consideration. If the decomposition of the culture-base of the feces continues for a length of time in a certain direction, there develop alongside of the coli bacilli characteristic vegetative formations (38) of which I shall here very briefly outline the following:—

1. Intestinal disturbances originating in the stomach (see VI A): bacilli of lactic acid, yeast, sarcina, flagellates, etc.
2. Fermenting stools rich in remains of carbohydrates (see VII 2. A): thread-like fungi (turning blue in iodine preparation) containing granules, spores, clostridia[1], bacteria and cocci.

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[1] These were recently cultivated by my assistant, *Dr. Kemp,* and he found them to be identical with *Grasberger's* granulobacillus butyricus.

3. Putrefying stools (see VII, 1. B): streptococci, proteus, pyocyaneus, bacillus bifidus, etc.

Of course, not all of these mentioned here need be present even under favorable conditions; now the one microbe preponderates, then the other, and is sometimes present in such abundance that it becomes conspicuous in the original microscopic slide or at any rate in the iodine preparation.

We can go even further in fixing the limits of certain vegetative formations. Thus *R. Schmidt* (38) speaks of a "pseudocolitype" in diseases of the appendix and the colon. I am of the opinion, however, that too great a specialization of the vegetative types is at present untimely, and I would reserve the culture process for rare cases of long-continued intestinal disturbances that cannot be classified among the known forms of intestinal disease or discoverable by the functional test.

At the conclusion of this section I might again point out that the fecal investigations here described—presupposing some practice—*as a rule,* do not require more than ten minutes. Next day nothing remains but the brief inspection of the incubator test and the sublimate test. For the most part I make all the tests one after another, with perhaps the exception of the test for dissolved albumin. In the repetition of the examination we can, of course, restrict ourselves to those tests which have furnished pathologic results. It is important to repeat the tests in all cases which are not entirely clear, for here conditions may change from day to day, just as in the case of the investigation of the stomach-contents. *Just in this fact, that the fecal investigation by means of the test-diet, shows the ephemeral variations of digestion which are lost in experiments of digestion, I see one of the chief advantages of this method.* All well know how quickly diarrhea and constipation, good and bad digestion, can follow each other. I do not understand, therefore, how it is that several investigators, who have employed our tests, have concluded, from the fact that the results vary, that our method is useless. If we wish to find objective symptoms for the lighter disturbances of digestion, which through our former inadequate methods of investigation exhausted themselves in subjective feelings, the test-diet must be administered often for a longer period of time and the fecal investigations made daily. The temporary pathologic conditions (fermentation, mucus, connective-tissue-remains, etc.) point out often enough that something indeed is out of order, and wherein that something consists.

The longer I work with the test-diet and these simple methods of investigation, the more indispensable have they become to me, and I do not doubt but that this would be the experience of every one who would adopt them with an unprejudiced mind.

IV. THE SEMEIOTIC SIGNIFICANCE OF PATHOLOGIC FINDINGS IN THE FECES.

Although this treatise is concerned principally with the functional examination of the intestine, *i.e.,* with the question why certain food-remains in a given instance pass through undigested, we must include in our investigations other pathologic findings in the feces, above all mucus, unchanged bile pigment, and the phenomena of fermentation or putrefaction.

1. Mucus.

The appearance of mucus in the feces indicates the existence of an inflammatory condition of the mucous membrane. It is the sole reliable indication of this. To this rule there are only two exceptions, and even these do not always hold: a thin mucous coating which, on drying, looks like varnish, is occasionally found on the surface of scybala in difficult defecation, without there being a true catarrh of the rectal mucous membrane; and in the so-called mucous colic, in which spasmodically large masses of glassy mucus are discharged, the nervous overproduction of mucus exceeds the local changes to such an extent that the aspect of the disorder eliminates the possibility of ordinary catarrh [*Nothnagel* (33)].

For the origin of the mucus and its common diagnostic application, the following is of importance:—

(a) With the exception of few instances the mucus that appears in the feces originates in the large intestine,

and it is supposed the higher the parts attacked by the inflammation, the smaller are the individual flakes and the more uniformly distributed are they found in the excrement.

(b) The assumption that it originates in the small intestine is admissible only in case of very fluid feces and only for the smallest mucous flakes, if these on microscopic examination are thickly interspersed with bacteria and half digested cells, or cell nuclei in characteristic arrangement. Bilirubin coloring of the mucous flakes (sublimate test), or the presence of fine bilirubin crystals under high power, furthermore the simultaneous appearance of the food-remains, especially that of muscle fibers and potato-remains (typhoid fever, tuberculosis, cholera, severe acute catarrhs), corroborate the above.

(c) The more thickly the mucus (in the microscopic preparation) is interspersed with cells (especially round cells)—whitish coloring can also arise if fat is enclosed—the more intense is the inflamed condition of the mucous membrane. In ulcerative processes there is found almost always only purulent mucus in the feces, scarcely ever pure pus.

(d) Ulcers and catarrhs, especially if they are high up, might exist without an evacuation of simple or purulent mucus or blood. The products of the inflammation are then dissolved by the digestive fluids just as the food ingredients.

2. Unchanged Bile Pigment (Bilirubin) and Variations in the Hydrobilirubin Content.

Normally after the passage of the intestinal-content through the ileo-cecal valve the bile pigment is, through the processes of reduction, connected with the putrefaction that here sets in, converted into hydrobilirubin. The hydrobilirubin assumes a red color in the sublimate-test, unchanged bilirubin becomes green.

(a) A green coloring of the entire excrement or of separate parts of it (chiefly of the muscle fragments and of the mucus) is—aside from the feces of newly-born babes or sucklings that scarcely need to be considered here—pathologic. It indicates a too rapid passage of the intestinal-content or a defect of the normal processes of reduction for other and as yet unknown reasons. Usually when the entire stool is colored green by $HgCl_2$ the small intestine also is affected. The diagnosis that the small intestine is affected becomes certain only in connection with other signs, as characteristic mucous flakes (see 1*b*) [*Schorlemmer* (39)], and undissolved food-remains (muscle- and potato-remains).

(b) The sooner after evacuation the feces are treated with $HgCl_2$ and the brighter their color is, the more intense is the red coloring (leuco-product). If the same phenommenon appears incompletely when the feces are fresh, then in most cases abnormal processes of decomposition are present.

(c) Complete absence of the red (and green) coloring in the sublimate-test occurs in fatty stools, when the bile is completely shut off from the intestines (the $HgCl_2$-test proves the complete obstruction of the biliary duct), and in rare instances in the so-called clay-colored stools without icterus (temporary cessation of the secretion of bile?).

3. Insufficient Digestion of Fat.

To answer the question, whether in a given instance the fat has been sufficiently digested or not, we have no uniform standard even when we use the test-diet. Inasmuch as every stool, even the fasting stool, contains fat—the normal excrement, about 23 per cent. of the dry substance—and inasmuch as too great a pathologic significance is not to be attributed to small variations of fat-digestion, it is only a *considerable* increase in the fat in the feces that must be considered practically.

This, however, is comparatively easy to recognize in macroscopic investigations, by the clayey appearance and the formation of a membrane on the surface, while the feces are being ground up with water, and in the macroscopic investigation by the large increase of flakes of fatty acids in heated acetic acid preparation (Preparation 2), and in some cases by the presence of drops of neutral fat or of fatty acid and soap needles in the native preparation. Fatty stools are of a light or whitish color, abundant and of acid reaction. I do not believe that a real fatty stool is overlooked easily, and therefore do not regard as necessary a further test for its recognition.

Fatty stools may occur:—

(a) In deficient secretion or outflow of bile. In this case there appears in the sublimate-test little or no red coloring of the feces. Usually icterus exists at the same time.

(b) In the disturbances of the pancreatic secretion. In such cases the fat frequently appears in the form of drops (neutral fat), and at the same time muscle-remains, recognizable macroscopically, are found in the feces. Hydrobilirubin represents, unless the flow of bile is checked, a somewhat frequent combination.

(c) In disturbances of the intestinal digestion. This is comparatively rare, at any rate it is not a correct rule that in intestinal disorders the fat absorption suffers the most. In extreme intestinal tuberculosis, intestinal amyloid, tabes mesaraica, and similar conditions, it is true, the fat absorption suffers more than the albumin digestion [*Weintraud* (40)], but a constant inter-relation does not occur here. Purely functional disturbances of fat digestion occur [*Salomon* (41)]. The recognition of all these conditions is possible only through the exclusion of *a* and *b,* while at the same time

attending to other pathologic signs (mucus, processes of decomposition, etc.).

After all, the fact that the improvement sets in after pancreatic preparations have been administered for an intestinal affection, may be of value. This will be discussed later.

4. Meat-Remains.

Through special experiments I established some time ago (42), that if 100 Gm. of beef, cut up sufficiently fine, are taken, under normal conditions, no macroscopically recognizable meat-remains reappear; whereas, they do appear in disturbances of digestion. On this is based the "meat test" interwoven into the test-diet, which, as already mentioned, was recommended as a special test by *Zweig* (27) and *Strauss* (28). *Roux* and *Riva* (43) "discovered" it anew in 1906. If care is taken to have the beef chopped sufficiently fine, and if, *at least in part, it is left raw,* these rules of diagnosis follow:—

(a) *If connective tissue-remains appear in the feces, it is a sign of disturbance in stomach digestion.* Of all the digestive secretions, as I have shown, the stomach juice alone can digest *raw* connective tissue.[1] The stomach alone is at fault if connective tissue appears in the feces. In most cases it is a question of hypochylia or achylia (as an independent disorder, in catarrhs, the development of cancer, etc.), and the disturbance may become so severe that the connective tissue is quantitatively discharged as a fine down intermingled with the whole stool. In many of these conditions probably an increased motility, too, plays a part, for in the case of normal chemic reaction, connective tissue may sometimes appear in the feces. Even in hyperacidity remains of connective tissue are found occasionally, and then, if the motility is normal or retarded, we must go back for explanation to an insufficient secretion of pepsin, which, according to recent investigation, may well be combined with increased HCl-secretion (44). At any rate, the disturbances of the activity of the stomach, which are indicated by remains of connective tissue, are not uniform. On the other hand, the reaction of connective tissue is extremely valuable in many instances and has often led me in the right direction. To this I owe the discovery that many more intestinal disorders than are generally supposed have their starting point in the stomach (45). This will be taken up later.

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[1] The statement of *Einhorn* (Arch. f. Verdauungskrankh., 13, '07, p. 475) that trypsin too can slowly digest connective tissue, I consider erroneous. Pancreatic digestion is by far the best medium to prove the presence of the connective tissue.

(b) *If macroscopic muscle-remains appear in the feces, this is a sign of a disturbance of digestion in the small intestine.* The stomach shares in the solution of muscle only to a slight percentage. By far the largest part falls to the share of the intestine. This is proved by the fact that even in cases of complete achylia muscle fragments will never appear in the feces. If the small intestine does not perform its functions normally, whether it be that the pancreatic juices are not sufficiently secreted, or that the active enterokinase of the secretions of the small intestine is absent, or further, that the highly increased peristalsis does not allow time for digestion, or, finally, that a primary disturbance of absorption leads to decomposition and thereby secondarily to diarrhea, the muscle fragments may reappear. *It follows from the above that the disturbance of a definite function of the intestine cannot be inferred from the appearance of muscle fragments alone. Even though difficulty in secretion must always be thought of* *first, we might still say, with certainty, that the disturbance is located in the small intestine,* for the large intestine is only a reservoir for after-digestion, and in it the solution of pieces of meat no longer occurs (46).

If the particular disturbance is not so severe as to cause the macroscopic muscle-remains to re-appear, an increase of microscopic muscle-fibers similarly denotes, under certain circumstances, a digestive disorder. Conclusions which are based on microscopic investigation alone are therefore to be used with caution. Nevertheless I have myself seen intestinal disturbances, which manifested themselves *exclusively* by a large increase of microscopic muscle-remains.

(c) *Remains of connective tissue and muscle alongside each other or together as meat-remains, indicate disturbances in which stomach and small intestine participate.* In an acute form this meat lientery sometimes appears in acute gastro-intestinal catarrhs. Through *Mr. Brink* (47) I have had such cases described, and I will later revert to them.

5. Carbohydrate-Remains (Fermentative or Incubator Test).

Of the carbohydrates contained in the test-diet, milk-sugar and white bread—leaving out of account very rare severe disturbances—are absorbed without any residue. The potato broth, accordingly, serves in the main as a criterion of satisfactory starch digestion. Normally, after eating the usual amount (one portion), there appears in the feces only a small number of empty potato cells (taking on a weak violet color, when stained with iodine). If the starch digestion is interfered with, a phenomenon which occurs even as an isolated disturbance (intestinal fermentative dyspepsia), the following combination is generally found: soft, pappy-consistency, a light brown, foamy appearance of the excrement, acid reaction, small microscopic and tapioca-like potato-remains, or only numerous microscopic potato cells with a content that takes on a blue color when treated with iodine, and granuliferous microbes of different sorts, the positive result of the fermentation test. Lighter grades of disturbed starch digestion show themselves through only a part of these symptoms. *Defective starch digestion has its seat in the small intestine; it depends most likely upon a disturbance of the secretion of the intestinal juice and* per se *(without any other alteration of the condition of the feces) signifies no severe disturbance* (26). Only the *positive* result of the fermentation test is of value for diagnostic purposes. The conclusion, that in the absence of the formation of gas, the digestion of starch is normal, is not admissible. For it may occur that the carbohydrate fermentation in the incubator-test may exhaust itself in the formation of acid products (without at the same time forming gas), *e.g.*, in fatty stools; it may happen that the carbohydrate fermentation is overtaken by processes of putrefaction that take place simultaneously (48, 49). Under certain circumstances the microscopic finding alone furnishes sufficient proof (numerous potato cells with iodine reaction, granuliferous microbes).

6. Relation of Fermentation to Putrefaction.

(Course of the processes of decomposition in the incubator test.)

The appearance of acid fermentation (C-H fermentation) is not the only alteration which the feces may undergo in the incubator-test. Sometimes gas also forms with an increasing alkaline reaction; the color of the excrement then becomes darker, the smell intensely putrid (albumin fermentation). In general, in carbohydrate fermentation gas is formed more rapidly and in larger quantities ("quick fermentation") ("Frühgärung"); in albumin fermentation it is formed later in smaller quantities ("slow fermentation") ("Spätgärung") (50). Slight degrees of one as well as of the other form occur even under normal conditions, though in the incubator-test the "normal excrement" shows practically no changes; *only higher degrees are pathologic.* The limit of one-half of the height of water in the ascension-pipe *c* of the fermentation tube has been determined empirically; it is not to be taken absolutely. Pronounced changes of reaction to one or the other side can under certain circumstances be just as important for judging the processes of decomposition as the gas formation. Especially to be considered as further characteristics for putrefaction are, definite alkaline reaction and odor of putrefaction in the freshly evacuated feces, a darker coloring, and the appearance of coffin-lid crystals (see above).

The manifestation of definite albumin putrefaction in the incubator-test allows us to infer severe disturbances of digestion, disturbances which are for the most part connected with anatomic changes in the intestinal mucous membrane. The cause lies in this, that the pathologic secretions of the intestinal wall (mucus, serum, pus), when mixed with the feces, are especially liable to decomposition.[1] That is why strongly putrefactive stools in diarrhea often afford a positive albumin-test (see Section III, 4). As *Schlössmann* (37) shows, in case of adults, dissolved albumin rarely passes from the food into the feces. More about this later (Section VII).

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[1] Perhaps because they are usually the carriers of special putrefactive excitants.

Experience, on the contrary, teaches that where albumin is imperfectly digested for purely *functional* reasons, higher degrees of fecal putrefaction appear rarely. The albumin-test is in this case, as a rule, negative (37).

The fermentation-test forms only one part of the incubator test which—speaking generally—tells us about the course of the processes of decomposition in the feces. *This orientation is, however, present only, when the one or other process (C-H fermentation or putrefaction) predominates decidedly. Where such is not the case, where a combination of both processes exists, we must for the present suspend judgment.*[1]

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[1] The account of the "fermentation test" and of its diagnostic signification here given differs in some respects from our original conception. I have assured myself more and more in the course of time that it possesses a more general value than merely to indicate a possible diminution of starch digestion. The assumption, that the "fermentation test" represents a universal test of the feces, which indicates even the mildest digestive disturbances, has led some investigators who occupied themselves with it after us, to entirely false conceptions (51). At no time have our statements given rise to such a misapprehension.

Since in the "incubator test," as I again desire to emphasize, only the *decided* changes toward the one or other direction (acid fermentation, putrefaction) are turned to account diagnostically, and an exact limit between normal and pathologic can indeed scarcely ever be established, I have given up altogether the weighing of a definite quantity of feces and instead have placed the limit of gas formation somewhat higher (one-half of the height of the ascension-pipe in place of the earlier one-fourth). I therefore lay the greatest importance on the simultaneous change of reaction (acidity of the excrement). In doubtful cases repeat the test with the next evacuation. For the experienced the changes in reaction alone can assume a meaning; nevertheless one should bear in mind that fatty stools also show acid reaction (on account of the abundant content of free fatty acids). In *putrefaction* the height of gas formation is even less significant than the other indicants.

V. COMPLEMENTARY METHODS AND FURTHER PROBLEMS OF THE FUNCTIONAL EXAMINATION.

What is attained?—Test of Mobility.—Tests of the Pancreatic Secretion.

It would be rash to assume that the methods which have been described in the preceding sections, the test-diet and the fecal examination based upon them, are at present adequate for a satisfactory functional examination. The goal before us, the obtaining of a functional examination analogous to that of the stomach, is still in the distance. *What we have attained is, first of all, a sharper demarcation of the diseases of the intestine originating in organic changes from the purely functional disturbances of its activity,* as far as the two can be separated at all. That this cannot always be carried out successfully is well known, and is based on the fact that a prolonged interruption of function eventually causes an irritation of the mucous membrane, and consequently catarrhs, in the same manner as a primary catarrh in most cases injures the digestion. The organic disease of the intestines manifests itself (apart from blood and pus), first of all, by the presence of mucus in the feces and, where this is absent, by a decided inclination to putrefaction and by a positive result of the albumin-test.

In the second place, the functional test settles the question which was formerly so difficult, namely, whether in a given case we deal with the affection of the large intestine or the small intestine, and to what extent the stomach is interested in the difficulty. This, however, does not imply that the former methods have now become superfluous. On the contrary, I consider it indispensable to supplement the functional test of the intestines—if there are no obstacles in the way—with a thorough examination of the stomach, and eventually also with the familiar methods of examining the rectum (rectroscopy, colonic washing, and inflation).

Finally, the functional test furnishes a sharp differentiation of the digestive power of the small intestine, according to the groups of foods (meat, fat, starch) to which the disturbance in the special instance relates.

These services are of great help, at any rate, if we consider how unclear, as yet, our knowledge is of the diseases of the small intestine (in the largest sense of the word, inclusive of the large digestive glands).

The next task would be to undertake a separate treatment in the sphere of the functional diseases of the small intestine, according to the three chief functions of the organ: secretion, motility and absorption.

The difficulty of this task lies more in the conditions of the intestine than of the stomach, where the three functions—in fact there are two, as absorption is not to be considered—do not so subtly interact.

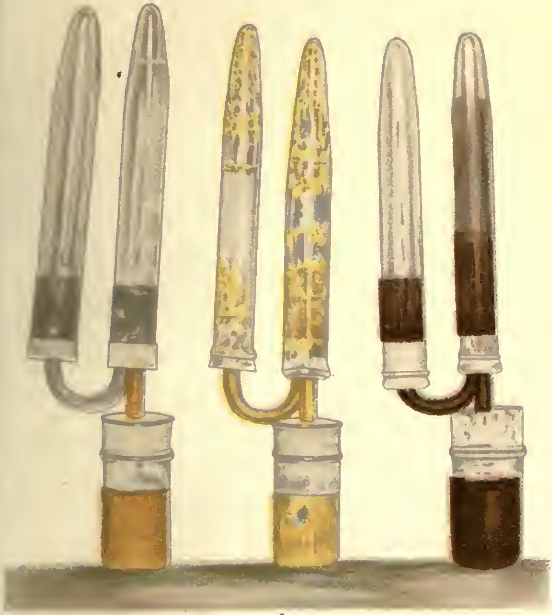
It holds true of the intestine that a disturbance of secretion must, in each instance, entail a disturbance of absorption; in fact, we conclude the former only by reason of the latter. The increased peristalsis also leads to disturbance of secretion. Such being the case, it is extremely difficult to separate the *primary disturbances of secretion* (be they functional or conditioned by the derangement of the absorbing passages) from the secondary ones. Fortunately the primary disturbances of absorption are rare, whereas primary disturbances of secretion and motility are frequent, and *we may, therefore, content ourselves to make their diagnosis by exclusion.*

As to the relation between the disturbances of the secretion and those of motility in the small intestine, it may be said in general that a disturbance of one of these functions does not necessarily involve a disturbance of the other. Pure disturbances of secretions in the bile, in the pancreas, and in the glands of the small intestine can exist without diarrhea; it is only the gradually developed decomposition of the undigested food-remains, particularly of starches and of albumin, which incite peristalsis.

For the length of time the food remains in the entire digestive canal—and this is the only sure guide we have at present by which *we judge motility*—the large intestine is, as is known, of extreme importance. In this the food-remains stay, on the average, fourteen hours, while they go through the small intestine, which is five times longer, rapidly in two and one-half to four hours. From the *passage-time of the ingesta,* therefore, we can judge in most cases only of the motility of the large intestine, and for this reason I consider the regular application of carmin in the test-diet superfluous (see page 14). We must strive to find a method of measuring the length of time the food remains in the small intestine, since the rapidity of the passage in the small intestine is no doubt of much greater influence upon the digestion than is the time the ingesta are present in the large intestine, in which, properly speaking, there is no more "digestion." If the small intestine alone is diseased, the passage-time can be appreciably diminished without diminishing the entire length of time (until the evacuation).

I regret to say that my efforts, extending over many years, to find a simple method by which to determine the time when the ingesta reach the ileocecal valve, have been without result. The experiments were discussed in the first

Plate III

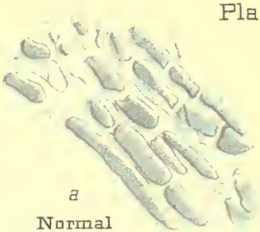


a
Normal

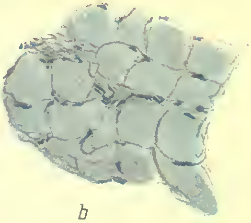
b
Carbohydrate fermentation

c
Putrefaction

Fig. 10. Incubator Test



a
Normal
No granules
Loose cellulose



b
Pathologic
granules present

Fig. 11. Granule Test.

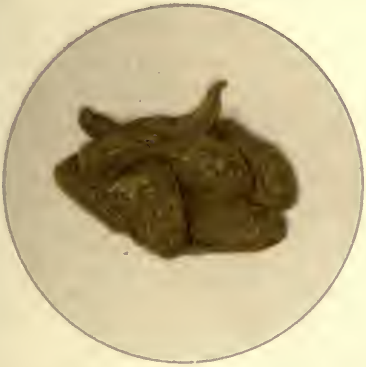


Fig. 12. Normal Feces

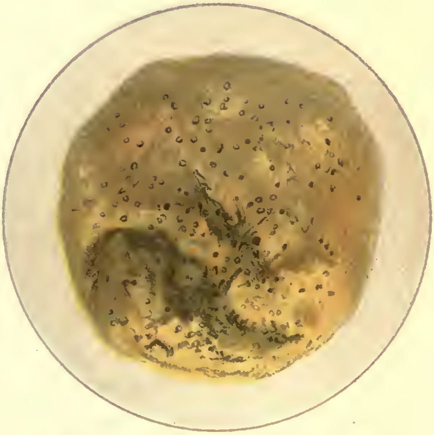


Fig. 13. Fermented Feces

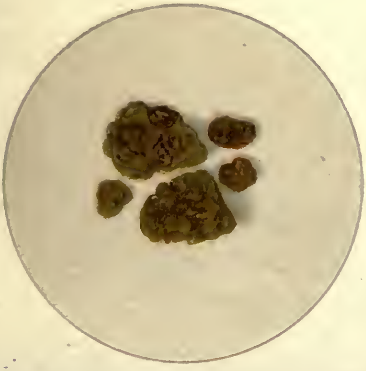


Fig. 14. Constipation Feces

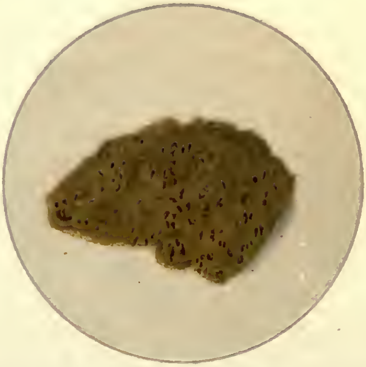


Fig. 15. Constipation Feces

edition of this book, and need not be spoken of here. The only means at our disposal now is transillumination by Röntgen-rays after prescribing bismuth-potato-broth, a process which was methodically worked out and tested by *Rieder* (52). In general practice, of course, it cannot be employed.

Under these circumstances we can hold, with some degree of certainty, the increased motility responsible for the re-appearance in the feces of undigested remains of the test-diet only when the peristalsis of the entire intestine is so much increased that the aggregate time of passage does not amount to more than a few hours, as occurs occasionally in cases of acute dyspepsia and catarrh (so-called jejunal diarrhea acc. to *Nothnagel*). But in all these conditions the secretory functions are always surely affected, and we will hardly be mistaken if we hold the secretory disturbance responsible whenever undigested food-remains re-appear in the feces.

In recognizing and differentiating the *disturbances of the secretion* we have advanced considerably, thanks to the test-diet, although here, too, the circumstances are by no means simple, for we possess several secretions for each group of food-stuffs (gastric juice and pancreatic juice for albumin; bile and pancreas for fat; saliva, pancreas, and intestine for starch). The stomach and pancreas (sometimes pancreas-intestine) can be separated through the meat test, as we saw in the preceding section. Fatty stools, from lack of bile and insufficiency of the pancreas, are comparatively easy to distinguish from each other, since in the two latter cases the digestion of meat, too, is generally affected. Concerning the re-appearance of starch remains, we may say that this is always due, according to the investigation of *Strasburger and myself* (13, 26), to the secretion of the small intestine, since we know from experience that neither the suppression of the salivary glands nor the stoppage of pancreatic secretion leads to a perceptible starch-lientery.

Nevertheless it is very desirable to obtain a direct method for recognizing the disturbances in the secretion of the most important of the digestive juices, the pancreatic secretion. For, by means of this, the recognition of disturbances in other secretions, as that of the bile and of the intestinal juices as well, is made considerably easier.

At present we possess three proposed methods to that end: the glutoid capsule-test of Sahli; my nuclei-test, and the urine-test of Cammidge. Let us examine these to see which has thus far produced the most satisfactory results.

1. *The glutoid capsule-test of Sahli* (53, 54) is based, as we know, on a peculiar hardening process of the gelatines. Gelatine capsules are treated for a varying period of time with formaldehyde vapors, whereby they are made proof against the digestive effect of the juice of the stomach, but not against the more powerful pancreatic juice (glutoid capsules). The capsules are filled with iodoform. As soon as the iodoform is set free in the intestine by the dissolution of the capsule, it is absorbed, and this point of time shows itself in the iodine reaction of the saliva. The difficulty with which *Sahli* had to contend in this method and which, according to my view, it will never be possible to overcome completely, is to strike the "degree of hardness" for the capsules, suitable for all cases. *For formalin has the property of making the tissue which has been impregnated with it proof against all digestion, tryptic as well as peptic.* This can easily be proved by test-tube experiments. Since the degree in which pancreatic digestion is stronger than peptic digestion is by no means a constant

one, but is subject to obviously large individual variations (according to the intensity of the secretion of the gastric and pancreatic juices; according to the length of time in the stomach, etc.), and, furthermore, since the preparation of capsules of a constant and uniform degree of hardness is scarcely possible, we cannot expect altogether uniform results even in the case of healthy persons. How much the less in the case of patients with failure of the gastric juice secretion, etc.!

Practical experience has unfortunately confirmed these fears. Already from *Sahli's* own numerous observations and still more definitely, from those of *Fromme* (55), *Galli* (56) and *Delachaux* (57), and the experiments made at my instance by *Wallenfang* (58), it appears that from a retarded glutoid capsule reaction the diagnosis of a disturbed function of the pancreas cannot always be made with certainty. On the other hand, the inference that a prompt reaction excludes the possibility of a disturbance in the pancreas, seems justified. *We can therefore attribute to Sahli's method at best only a negative diagnostic value, and that naturally reduces its value very considerably. It cannot be used, therefore, in practice.*

2. *My nuclei-test* (59) proceeds from a careful study of the varied behavior of tissue elements in the gastric and pancreatic juices (14). This showed itself, among other things, in that the nuclei of the cells, in direct contrast to the connective tissue, which was discussed above (page 23), are digested only by pancreatic secretions, and not by gastric juice. *If, then, undigested tissue nuclei reappear in the feces, we may safely conclude that there is an unsatisfactory functioning of the pancreas, just as similarly from the reappearance of connective tissue we may conclude that there is insufficiency of gastric digestion.* In

order to be able to find in the feces the piece of tissue which had been served, I have until now acted in the following manner: a small cube of meat[1] placed in a little silk gauze bag is served in a wafer with the test-diet, repeatedly for several days at noon. The little bags can easily be found again in the grinding of the excrement; they almost always contain small remains of the tissue. These are then rinsed off in water and examined either when fresh (with acetic acid or solution of methylene blue) or (in doubtful cases) after preparatory hardening and staining, for the presence of nuclei (see Fig. 11).

Einhorn (60) has recently proposed a technical modification of the nuclei-test by applying cubes of raw tissues of thymus, instead of beef. These tissues, rich in nuclei, have the advantage that we need not resort to hardening and staining. It is sufficient, if the regained piece of tissue is squeezed out upon a glass slide after a thorough rinsing, and, if nuclei are still found, they will be easily recognized in the juice of the tissue by the help of a microscope. Personally I have not as yet had any experience as to this modification, but I am not disinclined to accept it if it is of avail. I refuse, however, for reasons mentioned above, the further "improvement" of *Einhorn* of employing the tissue of thymus, tied to a bead, in the so-called bead-test (see page 19).

As to the practical use of the nuclei-test, the experiments on dogs, which *Wallenfang* (58) performed, have shown that, whereas no nuclei could be demonstrated in the meat-remains in the feces of the dog, after the complete extirpation of the pancreas *(after the complete extirpation only and not after the partial!)* the nuclei were preserved. From this method we may expect also practical results in man and the observations which I have (59, 61) thus far been able to collect seem to confirm this completely. These observations comprise nine cases, four of which could be controlled by section. *Wherever the pancreatic secretion was completely suspended, be it through functional obstacles or through organic diseases, the digestion of the nuclei, too, was suspended. If, on the other hand, the disturbance of the secretion was only partial, the nuclei were more or less digested.*

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[1] Fresh beef is cut in cubes about ½ centimeter thick and preserved in alcohol. After hardening, they are placed in very small gauze bags and again preserved in alcohol. Before using, the little bags must be well washed in water for several hours.

This limitation, that the nuclei-test leads to results only in case of *complete* insufficiency of secretion, is without much practical value, inasmuch as we know from numerous experimental and clinical observations that digestive disorders in pancreatic diseases appear only then, when the gland has entirely ceased to act. Important and significant, from a practical point of view, is the fact that under circumstances a too rapid as well as too slow a passage of the bag through the intestinal canal may deceive; the former because the nuclei may be preserved through the insufficient influence of the digestive juices, the latter because strong putrefaction may promote their destruction. Special experiments have taught me, however, that these errors are to be feared only when the entire time of passage is less than six or more than thirty hours.

Accordingly we will only be permitted to conclude with certainty from the reappearance of nuclei in the nuclei-test, that we deal with a pancreatic insufficiency, and a complete insufficiency when the time of passage of the bag comprises at least six hours.

Until now my method has not been tested very much.

Walko (62), in his investigations of pancreatitis, has declared it to be the best and the most reliable of all pancreatic tests; he believes, however—*and in this I agree with him*—that it is advisable to repeat it a number of times. *Einhorn* (60) likewise regards it as good; he fears, however—and his fear is based on the test-tube experiments—that in case of excessive acid secretion in the stomach or through the presence of abundant organic acids in the duodenum, the function of the pancreatic juice will not be complete. These doubts appear to be construed merely theoretically, for we know that hydrochloric acid is the strongest promoter of pancreatic secretion.

3. *The reaction of Cammidge* (63). This test, which is performed on the urine, is supposed to indicate pancreatic disease, and is based on the transition of certain products of disintegration in the pancreatic tissue (pentoses, nucleoproteid?) or upon derivatives of glycerin which were freed in the necrosis of the lardaceous tissue. According to the latest modifications it is carried out in the following manner:—

The urine—free from sugar and albumin—is carefully filtered; 20 cubic centimeters of the filtrate are slightly boiled for 10 minutes with 1 cubic centimeter HCl (specific gravity 1.16) on a sand-bath. A funnel, set over it, protects it against evaporating too rapidly. After this, the flask is cooled down thoroughly and is filled again with distilled water to a volume of 20 cubic centimeters. Add gradually 4 Gm. lead carbonate, let it stand several minutes and carefully filter it through a wetted filter until it is completely clarified. Add 4 Gm. pulverized tribasic lead acetate, shake it, and filter again after a short time until completely clarified. Remove the lead by adding 2 Gm. powdered sodium sulphate, heat until boiling, and cool down in running water to the lowest possible temperature. Filter off the white precipitate of $PbSO_4$.

Ten cubic centimeters of the clear filtrate are diluted up to 18 cubic centimeters with distilled water and mixed with 0.8

Gm. phenylhydrazin hydrochloride, 2 Gm. sodium acetate and 1 cubic centimeter 50% acetic acid. All this is placed in a small flask, with a funnel over it as a condenser, and is boiled ten minutes on the sand-bath, and while hot is passed through a filter, which was dampened in hot water, in a test-tube graduated to 15 cubic centimeters. If necessary, the filtrate is diluted with distilled water to a volume of 15 cubic centimeters.

After several hours (the test may sometimes last twelve hours) there will appear in case of a positive result of the reaction, light-yellow, flaky crystals, which, under the microscope, look like fine needles arranged in clusters and dissolve in 33% H_2SO_4.

The reaction is not easily carried out, nor easily judged, as other precipitates (of nuclei) may lead to error. The urine should be examined while fresh. The albumin that may be present must be previously removed by boiling with additional acetic acid. Likewise sugar that is present should be carefully removed by fermentation with yeast and by the expulsion of the alcohol through heating. A control-test with phenylhydrazin will show whether that is entirely removed.

I omit further discussion as to *Cammidge's* theory of reaction, since it is not yet sufficiently developed, and I confine myself to the question of its practical value. *Gruner* (64), who was the first to test it, found it negative in several indubitable cases of carcinoma pancreatis. *Haldane* (65) considers it entirely worthless and not characteristic. *Eichler* (66) found it positive in the case of three dogs on which artificial pancreatitis had been produced, but that it always failed in other cases. *Eloesser* (67) finally attributes to it a certain diagnostic value, but considers for the present extended experimentation very advisable.

As we see, the discussion as to Cammidge's reaction is not yet ended. Compared with the other two tests, it rests on entirely different presuppositions and, therefore, it cannot be properly looked upon as a characteristic of secretory insufficiency of the pancreas. Destruction of pancreatic tissue and even necrosis of lardaceous tissue can exist where there is no stoppage of the secretion of pancreatic juice; while, on the other hand, complete pancreatic achylia is possible in sound organic structure of the glands (61). We know also that the so-called "internal" secretion of the pancreas, which controls the metabolism of sugar, does not at all run parallel with the external secretion. The reaction of *Cammidge* can, therefore, well contribute to the diagnosis of pancreatic diseases—provided it gains further approval—but it does not give any direct information concerning the secretory function of the glands.

As a test of the secretion of the pancreatic juices, my nuclei-test comes into first consideration as one whose positive result has been repeatedly proved. If the nuclei-test shows a negative result, the glutoid capsule-test of *Sahli* may be resorted to for confirmation. If this is also negative, the secretory function of the pancreas is sufficient.

In the following there will be discussed a few, mostly functional diseases of the small intestine—for here the large intestine does hardly come into consideration—the delimitation and practicable treatment of which have become actually possible only through the systematic application of the test-diet. Of course the terse presentation given here does not claim the value of a clinical dissertation.

VI. INTESTINAL DISTURBANCES DUE TO THE STOMACH, THE LIVER AND THE PANCREAS.

A. Gastrogenic Intestinal Disorders.

THE view that a large number of the intestinal affections, especially the acute catarrhal and functional disturbances, are only a consequence of previous gastric affections is extremely suggestive, in view of the close coöperation of both organs; it is, indeed, very popular for the laity to ascribe a temporary diarrhea to a "spoiled stomach." It is a remarkable fact that this interconnection has for a time been altogether overlooked in medicine, and chiefly on the basis of *Von Noorden's* investigations (68), by which he proved that the digestion of food-stuffs can proceed normally in the case of patients with an increased or diminished or even completely lost secretion of the gastric glands, and that the metabolism of such patients did not *necessarily* undergo changes of any kind, provided that there are no high demands made upon the digestion [*v. Tabora* (69)]. Only recently has a change in this taken place: through *Einhorn* (70), *Oppler* (71), and various other investigators, it has been shown that there are diarrheas on one side and constipations on the other of purely gastric origin, and that the former are generally a consequence of diminished gastric secretion, especially of achylia; the latter (the constipation), on the contrary, are a result of hyperacid conditions. There are, however, not infrequently, some exceptions to this rule. *Schütz* (72), who has thoroughly investigated this subject, found extreme and protracted diarrheas even in cases of only slightly modified acid secretion and recognized their cause, among other things, in atonic conditions of the stomach, that is in reduced motility. The patients presented the appearance of a severe chronic and serious disease.

That these observations are not isolated, but that gastrogenic intestinal disturbances are *extremely frequent* and therefore have a great practical significance, I have repeatedly emphasized (73), and is now also acknowledged by most investigators (74). It is important to know not only that the underlying stomach disease may be of very diverse character; that it may sometimes have disappeared, and may not, therefore, be open to objective evidence; but that also the symptoms of the secondary intestinal disturbance may display an altogether different nature, *so that it is impossible to describe a specific symptom.* Usually we deal with diarrheas, which make their appearance as acute, chronic or also recidivous, and which, through a closer analysis, do not always appear as purely functional disturbances. On the contrary, they occasion the formation of severe catarrhs, which spread more or less extensively, and impress us at first sight as ulcerative processes (tuberculosis, dysentery). Even the pancreatic secretion may, through it, be affected sympathetically, so that the symptoms of the pancreatic affection are pronounced in the clinical status [*Schmidt* (61)]. On the other side we have constipation or constipation alternating with diarrhea, periodic discharges of mucus; in short, various symptoms suggesting disease of the large intestine.

The point in this matter is practically this, that in *all* cases of intestinal diseases, especially in affections of the small intestine, we must inquire whether they are not of a gastrogenic nature. *Now what evidences have we in answer to this question?*

I have established the following four diagnostic points of evidence:—

1. *Symptoms of an existing or previous stomach trouble.*
2. *The appearance of abundant connective-tissue in the feces after the test-diet.*
3. *The proof of the presence of specific microbes in the feces which often overwhelm its flora completely,*
    - (*a*) such as surely originate in the stomach and can often be, at the same time, verified as present in it (yeast, sarcinæ, long bacilli, flagellates),
    - (*b*) such as have propagated through the main course of the processes of decomposition (granuliferous microbes in fermentation, bacillus fluorescens, proteus, cocci, etc., in putrefaction).
4. *The prompt result of the therapy applied to the stomach.*

ad 1. *The underlying gastric affections,* as was already mentioned, may be of the most varied kinds. In the formation of secondary intestinal affections, hypoacidity and achylia lead; next come the atonic conditions and hyperacidity, and after these ulcer, carcinoma, and all other possible affections. If the condition of a tangible stomach trouble is in itself of importance for the gastrogenic nature of an undefined intestinal affection, it is particularly so to a high degree when processes of decomposition establish themselves in the contents of the stomach, whose irritants in that case (yeast, sarcinæ, lactic-acid bacilli) furnish characteristic vegetative products [R. *Schmidt* (38)]. But also in the case of complete normal condition of the stomach, it is possible that the stomach is responsible, if in the history previous stomach disturbances are reported, or if only a "weak" stomach is admitted. Let us bear in mind that some single germs of putrefaction and fermentation will always pass the digestive canal whenever occasion offers, but under normal conditions they are so affected by the stomach juice that their activity in the intestine becomes impossible. In order to multiply they must have passed the stomach under favoring conditions, at a time, therefore, when its activity was in some way disturbed. And after all, how little is necessary for a temporary derangement of the gastric digestion? I admit that, of course, for a permanent establishment of the microörganisms mentioned above, a single temporary insufficiency of the stomach is not enough. But the derangements may recur, and this usually causes a weakened intestinal digestion. This, however, is equivalent to a good opportunity for the propagation of germs. Still, I do not wish to anticipate the discussion of the pathogenetic relation.

ad 2. *The reappearance of macroscopically recognizable remains of connective-tissue in the stool after the test-diet is a simple as well as a reliable sign: it indicates that the stomach in the case in question was unable to perform the work which is expected of it.* This "connective-tissue-test," which I have firmly established after thorough and far-reaching experiments, both on sick and on healthy persons, is of greatest importance for the diagnosis of the gastrogenic disturbances of the intestine.

Of course, it can also be applied to the diagnosis of the stomach and for this special purpose *Sahli* has attempted to give it a simplified form in the desmoid-test.

The desmoid-test consists of tying a small rubber bag filled with methylene-blue with a thread made of catgut. This small bag is swallowed along with the noon meal. If within the course of the first twenty hours, the urine turns blue, *Sahli* concludes that the stomach has performed its work, for it is believed that gastric digestion alone is able to digest catgut. This would hold good, if raw catgut would conduct itself in regard to its digestion in exactly the same way as does the connective tissue of the chopped raw beef in the test-diet. But that is not the case. *Lewinsky* (76) has, upon my suggestion, carefully investigated this circumstance and found that differences exist, in so far as threads of raw catgut, if mixed with pancreatic juice and intestinal juice, will dissolve in 8-26 hours, while raw connective tissue—as I emphasize again in opposition to *Einhorn* (60)—will always remain unaffected. These differences are not at all surprising, as catgut is not pure connective-tissue, but is prepared from the *muscular stratum* of animal intestine. Therefore there arise also considerable differences if we compare the desmoid-test with the connective-tissue-test, and I cannot consent that the former be regarded as a substitute for the latter. While it may be useful for superficial information as to the gastric function, it is to be rejected for the diagnosis of the gastrogenic intestinal disturbances.

The reappearance of connective-tissue in the test-diet stools, recognizable macroscopically (apart from single tendon threads, which may be found in healthy persons as well), happens frequently; the stool may, in spite of this, have a healthy appearance, and need not, on further investigation, show any deviations from the normal feces. In rare cases I have found that the connective-tissue of the test-diet will be excreted again quantitatively, and then like felt-threads permeate the feces that are otherwise normal.

This circumstance points to the fact that not every disturbance of the stomach is *necessarily* followed by a disturbance of the intestinal digestion, not even when the former is very considerable. This agrees with the experience that only 15-20 per cent. of all the patients suffering from achylia are subject to secondary intestinal disturbances.

But this condition of the feces points also to another fact. If, by means of a tube, we examine the stomach of the patients that have connective-tissue in their feces, we do not find the expected achylia or hypacidity, in all cases. Some cases show entirely normal secretory conditions, others even hyperacidity. On the other hand, there are cases of complete achylia which, according to the result of the fecal examination, appear to be fully capable of digesting connective-tissue. It is natural in such cases to think of incongruities between the secretion of HCl and the production of pepsin, or that there are complications with motor disturbances (hypermotility). But even the consideration of these items does not by any means clear up all cases, and I must admit that I am in no position at present to adequately explain why certain stomachs which, according to the examination with the stomach tube perform their function faultlessly, do not digest raw connective-tissue, while others, although disturbed in the secretion, show themselves adequate to this task. The intestine acts here under no conditions by way of compensation. Whatever connective-tissue has passed the pylorus undigested, reaches the feces in that condition. Here, therefore, we are confronted with a still unsolved problem. It is certain that very slight disturbances of the complicated action of the stomach suffice to impair the dissolution of the connective-tissue, and accordingly *the connective-tissue-test may be regarded as an exceptionally fine criterion of the gastric digestion. It is not merely diagnostic of existent gastrogenic intestinal disturbances, but also an indication that such disturbances threaten to set in, even if they are absent at the time* when, perhaps, subjective feelings or intestinal irritations of short duration appear, which otherwise could safely be neglected.

ad 3. *The proof of the presence of specific microbes in the feces often overwhelming its flora completely*—which has largely to be done microscopically—is directly decisive for the gastrogenic origin of the disease only then, when the same microbes are present also in the contents of the stomach, or when the microbes in question are such as do not multiply in the intestine independently. This has been shown by *R. Schmidt* (38) in regard to yeast, sarcinæ, and the long bacilli. The propagation of the other decomposing agents (enumerated above under 3*b*) depends on the composition of the nutritive soil. Even if it is certain that they have immigrated through the stomach and their appearance presupposes a previous gastric disturbance, still there must be assumed a *secondary disturbance of the intestinal digestion* as a connective link between this gastric disturbance and their continued propagation. This disturbance of the intestinal digestion may, of course, be of diverse character. Most frequently we find symptoms of putrefaction in the case of gastrogenic diarrheas, and *v. Tabora* (74) speaks directly of an "intestinal putrefying dyspepsia" in opposition to the "intestinal fermental dyspepsia," which, in many ways, originates in the intestine primarily, as has been shown by *Strassburger* (26) and myself. He is of the opinion that the achylia of the stomach, injuring the digestion of albumin, supplies the intestine with abundant putrefiable matter, while hyperacidity impairs the digestion of carbohydrates, as is known, and thereby leads to the overloading of the intestine with fermentable carbohydrates. I cannot unreservedly agree with this opinion. I do not think that the disturbance of the gastric digestion is in itself sufficient to change the composition of the chyme so greatly; that happens only when the digestive secretions of the glands of the small intestine are secondarily impeded in their discharge, or when there is the concurrence of intestinal catarrhs. My investigations of functional pancreatic achylia (61) have made it very probable that long-continued achylia of the stomach may cause achylia of the pancreas, and I must agree with *Schütz* (72), when he speaks of a secondary insufficiency of the digestion of the small intestine. Doubtless there are also fermental dyspepsias of a gastric origin, in which cases the secretion of the glands of the small intestine must be assumed as secondarily injured. This last form leads to insufficiency of carbohydrate and, thereby, to fermentation, pancreas-achylia, and still more secondary catarrhs lead to putrefaction. It would lead too far to enter upon these matters more fully; I shall frequently return to them in the subsequent sections. Here I wish only to emphasize again *that the gastrogenic intestinal disturbances are of no uniform nature, not even as regards the course of the processes of decomposition: putrefaction occurs as well as fermentation, and each has its respective fecal flora.*

ad 4. *The result of a therapy applied to the stomach* is in the gastrogenic disturbances, in most cases, quite surprising. Of course this therapy must be adjusted entirely to the individual case, for the underlying diseases of the stomach may be of very diverse kinds. But even in unclear cases, which are not too rare, where the direct examination of the stomach affords no explanation, because secretion and motility perform their functions normally, and products of putrefaction are absent, and the feces, after the test-diet point through their content of connective-tissue, or the presence of certain microbes, to the stomach as their origin, an attempt in the treatment of the stomach should always be made, especially with the following means suggested by the conditions we have discussed:

(a) *Where hypacidity or achylia exists, hydrochloric acid must be given;* for lack of hydrochloric acid is chiefly responsible for the insufficient digestion of connective-tissue. I had the opportunity to convince myself in a number of cases that lientery of connective-tissue disappeared with the taking of hydrochloric acid, and along with it the intestinal complaints, at least, to a considerable extent. This experience corresponds with the investigations of *Leo* (77) made on the contents of the stomachs of persons suffering from achylia, which showed an essential improvement of the gastric digestion by the use of HCl. As regards the technic of the HCl medication, I prescribed, for the most part, until now, the pure dilute hydrochloric acid of the pharmacopeia in fairly large doses (30-50 drops with every meal); hereafter, however, I shall add pepsin to it, according to the investigations of *Leo.* He recommends particularly acidol pepsin tablets, strength No. 1.

(b) *As regards diet,* a general rule can be deduced from the lientery of connective-tissue along with the special indications of each individual case, which may be summed up in the rule: *Remove raw, half-raw, and smoked meat from the diet in cases of gastric and intestinal disturbances!* This rule, the reason of which I have discussed in detail elsewhere (78), is obvious from the foregoing. We have seen that the intestine cannot digest raw connective-tissue at all; it would therefore be foolish to offer it to a diseased intestine. We have seen further that an inability to digest connective-tissue manifests itself very readily in stomach troubles, that is, in stomach troubles of the most varied kinds, so that this fact forms a delicate test for this purpose. We would do well, therefore, to avoid raw meat altogether here, rather than to expose the patient with a disordered stomach to the danger of secondary intestinal disturbances.

From this rule we should deviate only when raw or smoked meat offers such corresponding essential advantages over meat cooked or well roasted that we are obliged to take the concomitant disadvantages along with it. How about this?

To be sure, the view that raw meat is especially easy to digest is firmly rooted in the popular mind, and there is a great number of physicians who believe they can support this view on scientific grounds. The reasons which they adduce are the two following: that raw meat stimulates secretion and possesses a great affinity for acids. The first of these arguments has been proved by the well-known and interesting experiments on animals by *Pawlow* (79); it holds especially in cases of sub-acidity or anacidity of the stomach. But if we compare the advantages which can be secured for the HCl secretion by serving raw meat, with the disadvantages that are caused by the connective-tissue which is introduced along with it, there cannot be any doubt but that the latter exceed considerably. This power of stimulating secretion has been proved only for the normally functioning stomach; in the case of a disordered stomach this power must certainly be very low. Moreover, it can be substituted in every case, as, again, *Pawlow* has shown, by meat extracts, bouillons, solutions of *Liebig's* meat extract, etc. The second argument, the great affinity for entering into combination with acids, is of importance in the treatment of hyperacidity. It contradicts to a certain extent the first-mentioned argument; for the question may justly be asked: Of what use is the increased affinity for acid, if at the same time there is an increase in the secretion of juices and *vice versa?* In cases of hyperacidity the conditions are such that any therapy which proceeds from the symptomatic indication to combine the abnormally formed acid, must yield to the more important *indicatio causalis* of that therapy which seeks to restrain the *formation* of acid. Avoidance of irritation and stimulation of the stomach is here the prime requisite! I have, therefore, long ago banished altogether from the therapy of hyperacidity all food-stuffs that irritate or excite secretion and I believe that I have benefited my patients more by this method than the so-called "albumin therapeutists" ("Eiweisstherapeuten"). If the acid must be neutralized, I prefer to administer some sodium, or to wash out the stomach, rather than fill the stomach again and thus never give it rest.

We see therefore that both arguments marshaled from the scientific standpoint in favor of raw meat, are not tenable. Only under one condition could raw meat be served, *i.e.,* if it could be freed from connective-tissue. In practice, however, this is almost impossible, even if the meat were chopped up or scraped ever so fine. It is a delusion to believe that scraped ham is free from connective-tissue. We need only try to remove the connective-tissue from ham or from raw meat as completely as the intestine does in case of achylia, and we will concede that this is impossible. The worst misunderstandings prevail in regard to smoked ham: smoked ham can never be ground up fine enough by the teeth, and yet there are people who do not consider it necessary to cut it up with the knife.

I will perhaps be confronted with practical instances and be told that we know a large number of patients whose nourishment we certainly could not have improved without raw meat as much as with it. It was the only thing they took in their severe sickness, and so on. Perhaps, further, the example of the English will be cited, who, as the layman believes, eat all their meat half-raw. Both are false. Appetite is not the sole criterion of how food agrees, else mushrooms, crabs, and nuts should be digested easily; the examination of the feces is undoubtedly a more trustworthy indication. And as regards the English, they never think of eating their meat half-raw, as is customary in North Germany; they eat it roasted rare, and that is quite a different thing. In meat roasted rare the connective-tissue is made dissoluble, in the half-raw meat it is not.[1] Therein lies the difference. In England raw meat is not prescribed for weak stomachs; that is only a custom in Germany and France.

(c) As a last and surest means the *lavage of the stomach* comes into consideration. I attained decisive results through it in so many stubborn cases that I recommend again applying it even in cases where there are no obvious reasons to regard the disease as originating in the stomach, namely, in unclear cases of chronic diarrhea, which *Schütz* designates as "chronic dyspeptic diarrheas." Ordinarily I combine with the lavage a wash of the gastric mucous membrane with salicylic acid or with silver nitrate, and I have repeatedly seen even desperate cases, such as had been treated in vain with all possible methods, cured through it. It is not too bold to argue from the success of the therapy what its probable cause is, that is, if we regard the diagnosis of gastrogenic diarrhea confirmed *ex juvantibus*.

A few words must still be said regarding the question how *the gastrogenic intestinal disturbances originate*, although it has been repeatedly touched upon before. While formerly the greatest value was attached to the absence of the disinfecting activity of the gastric hydrochloric acid, this fact can no longer be regarded as the only decisive one, since we have seen that also hyperacid conditions and motor derangements of the stomach may give occasion to secondary intestinal diseases. In the first edition of this work I, too, attached great importance to the lientery of the connective tissue, by calling attention to the fact that a bad dissolution of the connective-tissue leads necessarily to an insufficient metabolism of the meat and fat included in it (if it is not all chopped up), and that thereby a hiding place and a nidus is afforded to the decomposing microörganisms. Similarly *v. Tabora* (74) regards the inclination of the albuminous chyme to putrefaction as a most important point. Both are certainly correct, but we must equally regard some cases of fermental dyspepsia and other affections in which no connective-tissue reappears in the feces, as gastrogenic, and here this explanation involves us in contradictions. *Schütz* (72) explained the connection in this manner, that the increased demands, which in cases of insufficient activity of the stomach are put upon the digestion of the small intestine, lead gradually to an insufficiency of the latter. There is doubtless much truth in this, as the observations of functional pancreatic achylia (61) show.

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[1] Special experiments, with which *Dr. Goetz* is occupied in my laboratory, at present, lead to the view that the autolytic processes, which take place in the meat during "hanging," have a much greater share in the solubility of the connective tissue, than the cooking process. Entirely fresh connective tissue does not become more easily digestible through heat, not even through cooking; very slightly pickled meat, however, does.

I am of the opinion, at present, that all the items mentioned, work together; but that in one case one becomes conspicuous, in another case, another. The result in most cases is putrefying processes, which develop finally into catarrhs. In order that chronic putrefying processes may establish themselves in the small intestine, the condition of a suitable base must be present; besides, the respective decomposing microbes must be given an opportunity for lodgment. The absence or the insufficient activity of the gastric digestion may create both; the intestinal disturbances, however, gain independence only when there is added to them a secondary injury of the digestion of the small intestine (in the broadest sense of the word).

B. Hepatogenic Intestinal Disorders.

Significance of the Secretory Disturbances of the Bile for Digestion.—Influence of the Bile on Intestinal Putrefaction and on Peristalsis.

The hepatogenic intestinal disturbances are essentially conditioned by biliary obstruction. That increased secretion of the bile (pleiocholia or pleiochromia, *e.g.,* in loss of blood) also may result in intestinal disturbances is not clearly known. To be sure, in England it is customary to ascribe certain indefinite digestive disturbances (variable coloration of the feces, pressure, and fulness in the region of the liver, yellow coloration of the conjunctiva and the skin, and a general debility, etc.) to variations in the bile-producing action of the liver (biliousness); but these symptoms are too indefinite to admit of a pathogenic explanation, and therefore this doctrine has never been naturalized among us.

Biliary obstruction can be recognized usually by the white color of the feces, at any rate with certainty from the result of the sublimate-test; but there is an exception, the so-called "acholic stool without icterus," which is still an open question. *Nothnagel* (5), who first occupied himself with thorough investigation of these stools (where no *biliary obstruction* exists), formerly believed that they were to be explained by a temporary exhaustion of the biliary secretion, or by a functional hyposecretion. Later (33) he adopted the view of *v. Jaksch* (80) and *Fleischer* (81), according to which the cause is either a deficient fat absorption in the *intestine* or an excessive reduction of bilirubin (to leucohydrobilirubin). In the latter case the feces take on a brown color on the surface on exposure to the air; in the former case, when treated with ether. The sublimate-test is here, too, the most reliable; it never fails if hydrobilirubin or its reduction product is at all present. The latter produces, according to *Lifschütz* (82), a still nicer red color with a sublimate. However, when the stool is not subjected to an examination early enough or has already undergone a putrefying decomposition in the intestines, the tone of the color may become muddy-red or even brown-red.

In general we may say, therefore: Fluctuations in the amount of fecal coloring matter and also in its chemic constitution occur; but without biliary obstruction these are not sufficient to cause a functional disturbance of intestinal digestion—deficient fat absorption. Biliary obstruction, on the other hand, always occasions imperfect digestion of fat; indeed, in the complete cessation of bile (absence of all fecal coloring in the sublimate test) 25 to 50 per cent. of the fat of the food reappears in the excrement undigested. This well-known and seemingly simple phenomenon is, however, by no means fully explained. How does it happen that the pancreatic juice is incapable, by itself, of satisfactorily digesting the fat, especially since, according to *Volhard's* (83) investigations, the stomach already splits up the fat? According to recent physiological examinations, the effect of the bile is not limited to the forming of an emulsion and the stimulation of the intestinal epithelia for absorption, but it acts directly upon the steapsin. Still, we might expect that at least after an emulsified fat (milk) is presented, a still larger portion should be absorbed. We will, however, not enlarge upon these purely theoretical questions, but turn to a few *practical aspects* which are suggested by the absence of fat-digestion—fat-digestion alone, and not albumin- and carbohydrate-digestion being disturbed.

An old law still to be found in the more recent and even the latest text-books, says (in substance) that absence of bile in the intestine increases the putrefaction of ingesta and leads to constipation. This law is false, for the very opposite is true.

Whether the bile possesses such antiseptic qualities or not, the conditions in the intestine on which putrefaction of ingesta depends are quite different. The existence of an increased amount of ethereal sulphates and of indican in the urine discloses nothing in regard to the degree of intestinal putrefaction, as we shall see later. On this point only the incubator test gives us information and, if need be, the weight of the fecal bacteria according to *Strasburger* (84). These two methods prove, however, the absence of decomposition: fatty stools do not ferment, not even if starch is added. On the contrary they retard the fermentation. Neither do they putrefy; at the most fatty stools putrefy as a result of pancreatic and intestinal affections in which the albumin remains are at the same time increased. They react at any rate strongly acid, but that is the result of the large content of higher fatty acids, and it is these fatty acids which spread the bad odor. We can therefore perhaps speak of a decomposition of fat or rancidity, but not of albumin putrefaction. In regard to *the amount of bacteria* in the feces *Strasburger* found in icterus a striking diminution. *Hence, too, it is that icteric fatty stools scarcely ever cause catarrhs and inflammations of the intestinal mucous membrane.* This is of therapeutic importance, because from it we learn that the danger of occasioning intestinal disturbances through large amounts of fat in the foods does not exist *de facto*. Fat is not at all serious for patients with intestinal troubles if served in suitable form.

Constipation in patients with cessation of the bile I have never as yet met. In uncomplicated cases of cessation of bile the stool evacuation proceeds without retardation; indeed, on account of its soft consistency and the fact that the amount of the feces is about three times as large as the normal quantity, the stool is, on the contrary, surprisingly thin and frequent. This observation *Boas* (85) also has made; it is probably to be explained in part by the slightly stimulative action of the free fatty acids, present in considerable quantities.

C. Pancreatogenic Intestinal Disorders.

Significance of the Complete and Incomplete Absence of the Pancreatic Secretion for Digestion.—Diagnosis of Both Conditions.—Therapeutics.

If the pancreas is *completely* extirpated in experiments on animals, or if it is *completely* destroyed in persons, the following disturbances—aside from diabetes—occur: One-half of the albumin ingested is lost and similarly a large part[1] of the fat. In this connection it is a remarkable fact that here (in contrast to cessation of the bile) fat in emulsified form (milk) is better digested than unemulsified. With regard to the degree in which the fat splits, which according to the fundamental investigations of *Müller* (86) should be very much reduced, opinions are still at variance; the differences which have become known evidently come from the various kinds of ingested fats (emulsified or not), and perhaps from the varied intensity of the fat-splitting process in the stomach. The digestion of carbohydrates, as far as we at present know, is not considerably hindered by the destruction of the pancreas; here the intestinal juice steps in vicariously.

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[1] Writers here report very different amounts.

In the partial destruction of the pancreas, which occurs in man more frequently than the complete destruction, no disturbance of any kind need appear in the intestinal function. The same is the case with impediments of secretion by the contraction of the *ductus wirsungianus.* Just as pancreatic diabetes appears in experiments only after the last remnant of the gland has been extirpated, so the disturbances in digestion as a rule first become definite then. *I say "as a rule," for there are exceptions.* But the fact that considerable constriction, carcinoma—filtrations, softenings, and even apparently complete occlusion of the pancreatic duct by a stone can exist without disturbances of digestion necessarily following, is so striking that it deserves to be especially emphasized. I regard it as superfluous to establish this fact with references in literature and cases of patients; I refer to the well-known monographs (87) and to the practical experience which constantly confirms it for us. More important is the question, how is this to be explained? Is the compensatory functioning of small portions of the gland indeed so great that they can assume for a considerable period the function of the entire organ? That is not unlikely; for we know that of another organ likewise important for nutrition—the intestine—a third and more can be removed without the subject necessarily suffering in his nutrition. With regard to the liver, also, it is known that its chief function, the formation of urea, is disturbed only when scarcely any of the functioning parenchyma is present. Or does perhaps the intestine step in vicariously? Even that would be quite possible, for we know from the investigations of *Cohnheim, Glässner,* and others (88) that the intestinal juice is a synergist to the pancreas, in so far as its ferment, the enterokinase, is necessary for the action of the trypsin. Finally, for the cases of occlusion by stone, we must consider that the pancreas has almost constantly two ducts. It must also be noticed that the secretions may, perhaps, reach the intestines in roundabout ways through the circulation; that thus the external secretion is, so to speak, converted into an internal secretion. This is, of course, still entirely problematic.

But be that as it may, we must meet this condition. *For practice we learn from it, that whereas the complete destruction of the pancreas can be recognized without any difficulty, the diagnosis of an incomplete lesion of it is attended with the greatest difficulties.* In the first case we have again—in so far as intestinal disturbances are concerned—first of all a fatty stool. This fatty stool is differentiated from the fatty stool in acholia by various characteristics.

1. There is no absence of biliary pigment,
2. the fat is often (not always!) present as neutral fat (in form of drops under the microscope),
3. it contains at the same time always macroscopically recognizable muscle remains,
4. as a consequence of this complication, it sometimes shows putrefaction (either when fresh or after incubation). Fermentation also may occur.

On account of the phenomena of decomposition caused by the diminished digestion of the nutriment, the pancreatogenic digestive disturbances give rise to secondary catarrhs more readily than the hepatogenic, which, as already mentioned, can exist for months and even years without occasioning inflammation.

The diagnosis of *the incomplete pancreatic lesions* can in a few cases be made on the basis of the combinations of symptoms as here described, even if they are not very clearly marked, but this is rare. *We assume that the disturbance of the secretion affects the entire glandular tissue in the same manner,* so that not single parts of the gland, not even small ones, remain intact. This is the case especially in the degenerations and the proliferation of the connective tissue of the pancreas—the pancreas cirrhosis—the etiology of which is still unclear, and in the functional hypochylias or achylia of the pancreas. That such functional pancreas achylias, analogous to the gastric achylias, exist, I think (61) I have proved by the exact clinical analysis of three relevant cases.

The special tests for examination of the pancreas secretions, which have been discussed thoroughly (p. 50 ff.), *first of all the nuclei-test* and as their complement the glutoid capsule-test of *Sahli* and the urinary reaction of *Cammidge are of decisive significance for the diagnosis of these incomplete pancreatic lesions. The nuclei-test especially, if it show a positive result after repeated examination may, at the present stage of our knowledge, be considered as a proof of an insufficiency of the pancreatic secretion.* If it be negative and this result is confirmed by the glutoid capsule-test of *Sahli,* a disturbance of the secretive function of the pancreas may be excluded. True, the reaction of *Cammidge* does not admit a direct conclusion as to the secretive conditions of the gland; at all events it points to a degeneration of pancreatic tissues, according to another interpretation, to a necrosis of the lardaceous tissue, and serves therefore as an important aid for the diagnosis. Finally, the spontaneous or alimentary glycosuria gives us sometimes a valuable suggestion. We may hope, moreover, that the near future will evolve further "pancreatic tests," for it is of general interest and the possibilities are by no means exhausted. I refer only to the fact that according to the exact observations of *Deuscher* (89) lecithin is not subject to division but reappears as such in the feces, when the secretion of the pancreas is absent.

It is self-evident that the special pancreatic tests here mentioned retain their diagnostic value even in the case of *the complete destruction of the pancreas;* only they are as a rule not so necessary in this case, because the characteristic changes of the test-diet-stool become so conspicuous, that they alone make the diagnosis certain.

For the pathogeny and therapy of the disturbances of the pancreatic secretion very little that is tangible has been accomplished by the functional test. From the analysis of my cases of functional pancreatic achylia I have gained the conviction, that very probably they were produced secondarily by the stomach, so that in the last instance they belong to the great group of "gastrogenic" intestinal disturbances which, as we have seen, comprise affections of the intestine of diverse clinical character, and that the secondary "insufficiency of the digestion of the small intestine" must, in many ways, be considered as the connecting link between the stomach and the intestine. The consequence of this opinion in regard to therapeutics is, that such cases must be attacked by a systematic treatment of the stomach (HCl-medication, lavage of the stomach), and in fact I have attained success in this manner. Along with this the organotherapy, *i.e.,* the treatment with pankreon comes into consideration. It may, as *Salomon* (90) has shown under certain circumstances, greatly improve the metabolism of the food, thereby giving further welcome confirmation that the disturbance of absorption, as found, has a pancreatic origin.

VII. INDEPENDENT INTESTINAL DISTURBANCES.

The digestive disturbances inhering in the intestine itself are very numerous and manifold. An exhaustive presentation of them is naturally not intended here; only a few diagnostic, pathogenic, and therapeutic points, which have been obtained from the systematic application of the test-diet are here to be taken at random. The superscription: "Independent Intestinal Disturbances" is chosen, because the usual classification into primary and secondary intestinal affections easily gives rise to false notions. The independent intestinal disorders do not by any means always originate in the intestine. As we have seen, a gastrogenic intestinal digestive disturbance may gradually assume an independent character, and as such it may continue to exist even after the gastric disorder has terminated. Further, there are severe intestinal diseases which originate in the blood, as the diarrheas in uremia, pneumonia, septicemia, etc.

In the more important processes of infection of the intestine (typhoid fever, tuberculosis, cholera, dysentery) the point of entrance of the disorder is to be sought in the intestinal mucous membrane itself; still it is a question about which we are not as yet sufficiently informed, why the exciting causes of the disorder lodge just here and how it arises in individual cases. Presumably the stomach plays a part in this connection, for it is to be regarded as an established fact that in epidemics of typhoid fever and cholera, disturbances of gastric activity strongly predispose to infection.

1. Organic Disorders.

Relation of the Organic to the Functional Disorders of the Intestines.—Causes of Diarrheas in Cases of Ulcers.—Dietetic Inferences.—Catarrhal and Nervous Forms of Diarrhea.—Serous Transudation.—Diet in Chronic Catarrhs.—Salicylic Milk.—Astringents.—Intestinal Atrophy.

With regard to the independent disturbances of intestinal activity, in so far as they will concern us here, stress is to be laid on the fact that they *very seldom* run their course *as purely functional* disturbances, *i.e.,* without anatomic changes of the mucous membrane. We must therefore include ulcers and catarrhs within the sphere of our consideration. The relation of organic to functional disturbances is an intimate one, especially in the intestines, but it is at the same time so very variable that for the most part a clear understanding of it is to be obtained only with difficulty. As an example I take at random, first, the diarrheas in intestinal tuberculosis and intestinal typhoid.

A. ULCERS.

It is almost universally known that *diarrheas* form a frequent though by no means a constant symptom of typhoid fever, intestinal tuberculosis and of intestinal ulcers in general. *Nothnagel* (5) in his fundamental investigations has duly appreciated this fact. There are cases where, in spite of the intense ulceration of the intestinal wall, entirely normal stool, and even constipation exists. On the contrary, we occasionally see *intra vitam* severe, uncontrollable diarrheas, whereas, *post mortem,* the intestine shows only a little isolated ulcer or only a cicatrix. Instances of the one as well as of the other kind I have thoroughly investigated *post mortem* macroscopically and microscopically, and convinced myself that the mucous membrane, in so far as it has not collapsed under the ulcer, remains unchanged; and this is true even for the immediate neighborhood of the ulcer. *A concomitant diarrhea is sometimes absent in cases of ulcers.* I lay stress on "sometimes," for in other cases we find the mucous membrane more or less inflamed, and then the accompanying catarrh often comes to the front in the clinical picture of the disorder. In the examination with the test-diet we rarely fail then to find the little mucous flakes of golden yellow color containing bilirubin, which, judging from their microscopic characteristics, come from the small intestine. These flakes can come only, of course, from the mucous membrane itself and not from the base of the ulcer; they give us an important indication for the recognition of the accompanying catarrh and its localization. For the diagnosis of the ulcers themselves they are of no value. As a somewhat certain sign of the ulcerous degeneration, only the proof of the presence of pus and blood in the feces can be considered, and even this only qualifiedly. Pus may penetrate into the intestine from the surroundings, and blood may originate in the stomach or the esophagus. Pus, produced from the intestinal wall, moreover, is found undigested in the feces only when it originates in the lower part of the colon. Red, unchanged blood, is found in the feces only when it originates in the lower parts of the colon; most often it is due to hemorrhoids, polypi, and carcinomata. Blood from higher portions of the intestinal canal is transformed into hematin and in larger quantities it gives the familiar tar-color to the feces. Smaller (occult) admixtures of blood can be recognized only by aid of the test of *Weber.* Besides ulcers, congestive conditions also may lead to transfusion of blood. Because of the presence of the different products of secretion, to which the transudated serum also belongs, the feces incline to putrefaction in case of ulcers. Probably this inclination to putrefaction produces also the diarrheas. More about this under B.

The treatment of intestinal ulcers, especially of the tubercular, is a very thankless undertaking. No kind of dietetic regimen can here lay claim to general validity; indeed, we see frequently that very coarse diet, especially vegetables, agrees excellently (at least for a time) whereas, every bland diet causes uncontrollable diarrhea. In practice, in cases of intestinal phthisis, where after the test-diet the signs of catarrhal inflammation of the mucous membrane (mucus, putrefaction) are wanting, or are insignificant, in the excrement, we can always find justification from the above not to limit the diet too closely, but to vary the food prescribed, and occasionally even run some risk. In typhoid fever naturally we are more loath to do so; and yet I should like to express it as my personal conviction that under normal conditions of the stool such an exclusively liquid diet as is now customary is no postulate in typhoid fever. I myself would have no scruples, for instance, against softened zwieback, which is dissolved long before it reaches the intestinal ulcers, or against butter or broth made of potatoes and rice strained through a hair-sieve, or even against finely chopped meat freed as much as possible from connective tissue. Relapses we find occur as often among patients who have been under the strictest control as among careless private patients.

B. CATARRHS AND SEROUS TRANSUDATION.

The intestinal catarrhs, especially the lighter chronic forms, are sometimes combined with nervous irritation, and we often see, therefore, that the organic and nervous elements govern the course of the disease in varying degrees. On other mucous membranes, as in the respiratory tract, the stomach and the bladder, etc., we have likewise nervous symptoms complicated very generally by organic disturbances. The pertinently named "mucous colic" of *Nothnagel* resembles asthma so completely that both conditions have been compared in many respects with each other, and asthma is just the type of a "nervous catarrh" (91). The more cases of chronic diarrhea I see, the more am I convinced of the fact that many cases passing under the diagnosis of nervous diarrheas are slight chronic catarrhs, whose respective clinical pictures the nervous elements influence largely. Most clearly the combination of catarrh and nervous irritation confronts us in *colitis membranacea,* a process combined with a more or less abundant discharge of mucus, and located in the large intestine exclusively. Now it is stimuli going out from the genitalia, now psychic influences, and again sensitiveness to certain definite foods or temperature, even to the movements or position of the body, which are associated with often altogether insignificant and circumscribed conditions of inflammation in the mucous membrane. Analogous processes are naturally also in the small intestine. Here, too, as a rule, the exact functional examination will at once clear up the situation, inasmuch as the fecal examination will show either small mucous flakes or marked fecal putrefaction, which, as has been repeatedly emphasized (see page 42), is maintained chiefly through the admixture of soluble mucus or other inflammatory products from the mucous membrane.

To these inflammatory products belongs also the *transudated serum,* which, it seems, is easily secreted in the intestine, and, according to the result of the examination of the feces, mixes with the chyme, in various diarrheas, which, in many respects, are considered purely functional (as after purgatives, in case of entozoa, in the so-called toxic forms which proceed from the blood, and in some "nervous" forms). Of course, we cannot prove its presence as such in the feces, but the probability is very great that such a process of transudation has taken place, if the incubator-test shows a marked inclination of the feces to putrefaction and there is no mucus, blood, pus, or abundant remains of albumin present to explain this, and if the chemical examination, moreover, shows the presence of soluble albumin. Through the careful investigation of *Schlössmann* (37) it has been shown that in the stool of adults soluble albumin originates almost always from the intestinal wall and not from unabsorbed food-remains. In this regard it resembles the nucleoproteid of the feces, only the latter appears even in the normal excrement. We may regard the epithelia of the intestinal wall as the place of origin of the nucleoproteids, a certain percentage of which is always discharged into the lumen through the natural channel. Under pathologic conditions of the intestine we often find the nucleoproteid of the feces increased, yet we cannot draw any certain diagnostic conclusions from this, for it is difficult to draw the limit between normal and increased precipitate. The conclusion that there is a pathologic secretion of the intestinal wall is therefore more probable in case of the presence of albumin (page 31), for albumin never appears under normal conditions.

Albumin is found only in diarrheic feces, often together with mucus, and then it is difficult to decide how much of its presence is due to the albumin-components of the easily decomposing mucin. I can conceive that in case of a catarrh located exclusively in the small intestine no mucus, as such, is found in the feces, but only the products of its decomposition. Such a stool putrefies, and shows a reaction for albumin, but it discloses no mucus, and therefore cannot help in the diagnosis of "catarrh." As a rule, however, at least when it is a case of diarrhea, a few flakes of mucus will not be overlooked. Therefore it may be set down as a rule, *that a diarrheic stool, which contains no mucus* (and not too many macroscopic muscle-remains), *but shows a reaction for albumin and putrefies in the incubator-test, is mixed with transudate serum, and therefore, points to an irritated or inflammatory condition, or to ulcers of the mucous membrane.*

If we apply this rule to the quite numerous unclear or so-called nervous diarrheas, we shall, in most cases, gain the conviction that the mucous membrane of the intestine is not quite intact, and that, therefore, purely functional or purely nervous diarrheas are very rare. We must admit, of course, that there may exist a nervous diarrhea with transudation of serum, just as there exists a "nervous rhinitis." But, whether we are justified to speak in such cases of *"purely"* nervous diseases may be questioned. These cases can be best understood, if one conceives, that in a sensitive nervous system clinically severe diseases (diarrheas) may result from slight and even very slight affections of the mucous membrane.

The *therapy* of such processes must be instituted simultaneously at both ends, at the inflamed organ and at the nervous system. As one or the other component predominates, greater stress is to be laid on one or the other. We wish to consider here only the local treatment and here I would like to discuss briefly two points which I have learned as a result of the function-test. The one concerns the diet, the other medicinal therapy. The diet in intestinal catarrhs and serous transudations should be as unirritating as possible, easily digestible and non-putrefying. To meet these three requirements at once is indeed difficult: in the first place, the difficulty is to be approached with the old established gruels (schleimsuppen), provided that milk does not agree. It is necessary always to aim at accustoming the intestine to milk again, that food-stuff, which with high nutritive value claims relatively the smallest amount of digestive work—and this is best accomplished by adding gradually to the soups a constantly increasing quantity of milk. *The reason why the gruels, especially the carbohydrate food-stuffs, are to be preferred in intestinal catarrhs is a very simple one, they do not putrefy.* Wherever the carbohydrates and albumins are jointly subjected to decomposition, albumin putrefaction does not set in as long as there are present carbohydrates capable of fermentation; the carbohydrates to some extent shield the albumin (here the inflammatory product of the intestinal wall) from putrefaction. It is established beyond doubt that the milk diet furnishes the smallest amount of putrefactive products (92), and in that, too, it surpasses all other food-stuffs. Of course, we must reckon with the fact that with our diet, intestinal putrefaction suddenly passes over into intestinal fermentation which the incubator-test then shows. Obviously the irritating effect of the milk is restricted in this way. In itself this is not so bad, for fermentation is at any rate the lesser of the two evils; but we should try to prevent it, and the simplest and surest way of doing this is by the temporary addition of small amounts of salicylic acid to the milk (or also to the soups) in the following manner:

The daily portion of the milk, which is to be given the patient is boiled with 0.2 Gm. of pure salicylic acid (pro liter), first grinding up the required quantity of salicylic acid with some milk in a mortar, and when it is finely divided, adding it to the whole mass.

As *Strasburger* (84) has shown,—next to the reduction of the amount of nutrition—this is the best method to reduce the number of intestinal bacteria. On the basis of my experience I should like to express it as my opinion that salicylized milk has stood the test excellently, not only with adults, but also with children, where we unfortunately have to deal too often with severe intestinal catarrhs. It removes the injurious qualities of the milk without impairing its good qualities.

As regards medicaments, outside of opium, astringents are to be considered especially in chronic catarrhs. In the oft-quoted work of *Strasburger* it has been proved that these remedies diminish the bacterial content of the feces better and with more certainty than purgatives. Calomel, which is still regarded as the intestinal disinfectant κἀτ' ἐξοχὴν in fact increases the fecal bacteria, as is now established beyond a doubt (8, 9, 93). I find fault with the astringents because when administered in insoluble form (as tannigen, tannalbin, powdered bismuth) they may occasionally produce irritation on very sensitive persons. The inflamed intestine is extremely sensitive to all insoluble and undissolved substances (remains of cellulose, potatoes not divided fine enough, carmin powder, etc.). Systematic fecal examinations show this. Not only all the food substances should be given in soluble form, but also the medicines, and that is best achieved through the chemical combination of both. Therefore I give tannin, *eg.*, in the form of "milk-somatose" (somatose of milk albumin with 5 per cent. of chemically combined tannin), a preparation with which I have had useful results, even with very sensitive intestines (94).

I do not wish to enter more fully on the efforts to disinfect the intestine by the ingestion of suitable medicaments.

It is possible that results can be obtained by this means, but surely, only, if the conditions of the putrefaction are, at the same time, rendered worse. I am occupied with experiments in that direction.

C. INTESTINAL ATROPHY.

A few words more concerning the interconnection of long-continued intestinal catarrhs or putrefactive decomposition of the intestinal content with severe (pernicious) anemias may here find a place. On the basis of clinic and anatomic investigations, *Nothnagel* (5) years ago described the atrophy of the mucous membrane, *i.e.*, of the ileocecal region, as a frequent result of chronic catarrh and assigned to it a special place in clinical pathology. At first these investigations aroused but little interest, but as the atrophy of the gastric mucous membrane came to be better known, they were again taken up and the now widespread view developed that the atrophy of the mucous membrane of the stomach alone, as a rule, did not involve severe disturbances of the general condition of health, but that if the process extended into the intestinal mucosa, general disorders and, particularly, pernicious anemia could easily result from it. This view which obviously is based on the entozoön anemia, however, was, until recently, without basis in fact. All the more recent investigators, who have carefully studied the question of intestinal atrophy, and I myself am in accord with them (95), have had to convince themselves that an anatomic process which expresses itself as an atrophy of greater portions of the intestinal mucous membrane, is not to be found at all or only as an exception in pernicious anemia. The mucous membrane is usually normal, or at most, affected by a slight catarrh. At any rate, these circumstances are not specific, despite the assertions of *Ewald* and *Hansemann* (102). The symptoms on the basis of which *Nothnagel* founded his belief are obviously for the most part artificially produced, and *Nothnagel* (33), with the unselfishness of the true scholar, has admitted in the last edition of his "Erkrankungen des Darmes und des Peritoneums" (Diseases of the Intestine and Peritoneum), that he might have been mistaken. Also, the clinical fecal conditions, which *Nothnagel* described as characteristic of intestinal atrophy—an increased content of water with the absence of any other anomaly—when investigated with the test-diet scarcely occur; almost always, where there is an increased content of water, either products of inflammation (mucus, serum) or indications of a functional disturbance (undigested food-remains) are present at the same time. But in spite of this the theory of the enterogenic origin of pernicious anemia has persisted; it has merely assumed another form. *Grawitz* (96), who still defends it, supposes that, in absence of atrophy, it is possible that, in cases of protracted catarrhs, abnormal products of decomposition of the intestinal content exert a toxic influence on the blood (Blutgewebe). How many pernicious effects have been attributed to the abnormal products of the decomposition of the intestinal contents, and how little positive evidence has remained after the critical sifting by *Fr. Müller* and *Brieger* (97) of the autointoxications arising in the intestine!

If the patients who suffer with pernicious anemia are systematically examined by means of the stomach tube and the test-diet, it certainly appears that the majority of these suffer from achylia gastrica and often from the disorders of intestinal activity consequent upon it (diarrheas and catarrhs and increased putrefaction in the incubator test).

Exceptions occur, however, as *Einhorn* (98) especially has lately emphasized. But how many patients suffering with gastrogenic intestinal disorders are there, the condition of whose blood is altogether normal? I estimate their number so high, that in comparison the little group of pernicious anemias disappears altogether. Already this condition does not speak very well for a causal connection. Even less so does the chemical investigation of the products of putrefaction formed in the intestine which are found in the excreta of such patients. Hitherto we had at our disposal for the purpose of orientation as to the amount of enterogenic products of putrefaction only methods of demonstrating these in the urine. *Strauss* (99), who carefully investigated a pertinent case by means of these methods, could not convince himself of an excessive intestinal putrefaction. Through *Ehrlich's* aldehyde reaction (100) we are now able to estimate the amount of indol in the feces. *Baumstark* (101) has studied this reagent under my direction. He found in pernicious anemia an increased total amount of indol (feces plus urine), but the amount varied within the limits which occur in other cases of achylia gastrica.

Such was the state of things until recently. In order to suppose a specific intestinal putrefying or toxic process as the basis of a pernicious anemia, we had to have stronger proofs. French investigators (103) believed they had found such a proof in the poisonousness of the intestinal contents, especially of the contents of the small intestine. *Magnus-Alsleben* (104) has verified these facts and elaborated them, by demonstrating a toxic substance in the mucous membrane itself. But not much was gained even by this. *Tallquist* (105) showed very recently, that in the normal mucous membrane of the stomach and of the intestine there occurs a hemolytic lipoid-substance, which in its effect resembles exactly the toxin of the bothriocephalus latus. It is likely that in case of a diseased stomach or intestine, this substance gets into the blood in increased quantity and affects it destructively which, under normal conditions, is somehow counteracted. The future will show whether the investigations of *Tallquist* have created a point of departure for the understanding of the pernicious anemia.

2. Functional Disorders.

In spite of the intimate connection between organic and nervous affections of the intestine, which has been emphasized repeatedly in the preceding section, we can nevertheless mark off here a considerable number of different disorders of intestinal activity, which are exclusively or, more cautiously expressed, quite predominantly of a functional nature, and which we may therefore designate in accordance with the principle *a potiori fit denominatio* as functional disturbances of the intestine. *With the stomach, the heart, and sexual organs, the intestine is a favored organ for the localization of general nervous conditions.* In intestinal diseases the layman regards the kind of evacuation—diarrhea or constipation—as the most important sign, in accordance with which the entire disease is to be judged. From the physician's standpoint this conception is altogether fallacious. *One diarrhea is often entirely different from another diarrhea.* The conception of diarrhea implies a too thin consistency of the stool and a too frequent evacuation. Both elements are indeed usually combined with each other, but to a certain extent they may also appear independently of each other. Thus there occur just in nervous conditions very frequent dejections without essential modifications of the feces, and again, on the other hand, one daily evacuation of a thin stool. *Diarrhea is altogether too ambiguous a symptom to enable us to derive from it anything more than a suggestion for further investigation.* At any rate, it is necessary always to bear in mind that in proving the presence of a diarrhea—and the same holds with regard to constipation—a diagnosis has not yet been made. This can only be obtained on the basis of all the symptoms that present themselves or of a more protracted observation.

Even if we must admit that the motor function of the intestine in one way or another participates in most purely functional disorders, the systematic application of the test-diet nevertheless shows us not infrequently disorders of intestinal activity which must be regarded as purely or almost purely secretory or absorptive.

A. SECRETORY DISTURBANCES.

Intestinal Fermentative Dyspepsia.

Among the former, the independent *secretory* disturbances, I count the majority of cases of *intestinal fermentative dyspepsia,*[1] recognized by *Strasburger* and me through our function-test and described by us as a special form of disease. We were able to collect a rather large number of cases, where essentially subjective disorders existed: abdominal pains, borborygmus, diminished appetite, general malaise; whereas, on objective examination, the patients gave the impression of being in tolerably good health. Now and then the abdomen was somewhat swollen, perhaps diffusely tender or tender only in the umbilical region, the tongue was somewhat coated, defecation took place one to three times every day without real diarrhea.

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[1] In our detailed communication concerning this (26) we left the question as to the nature of the disease still open; still even at that time our thoughts inclined toward a functional disturbance, essentially secretory, of the digestion of the small intestine.

The examination of the feces after the test-diet proved that the usually mushy stool was filled with numerous gas bubbles, foamy, light yellow in color, showed an acid reaction, and had a very strong odor of butyric acid (see Fig. 13). In other cases these characteristics were absent, the stool appeared normal on macroscopic investigation; on microscopic examination, however, more or less abundant remains of starch-granules in the potato-cells and various kinds of granuliferous microbes became visible, most frequently those usually known as clostridia, which resemble the yeast-cells in form (see Fig. 8f.); further, long threads of a leptothrix character, and the like. In the incubation-test there always appeared also C-H (carbohydrate) fermentation. This fermentation was not always equally pronounced; it could greatly fluctuate in intensity from one day to the other, as there is in general no constant ratio between it and the quantity of the starch-remains in the feces (Cf. the remarks in Section IV, 6).

There could be no doubt that it was a case of insufficient starch digestion, for from what other source could fermentable matter get into the feces? This disturbance was isolated, for other signs of abnormal function (muscle-remains, etc.) were completely absent. Nor did the examination of the content of the stomach show much that was pathologic. The chemic analysis which we performed in eight cases also confirmed this assumption; we did not find the quantity of N (nitrogen) and fat in the feces greater than in normal stools, but, on the other hand, we found the C-H content almost doubled.

If, accordingly, the bile and the pancreas must have had their normal function, nothing remained but to attribute the cause to a disorder in the secretion of the succus entericus. Of the succus entericus we know that it can develop powerful amylolytic action and that this may extend into the large intestine. Certainly the disorder in secretion here also was in the foreground; in so far as the motility participated at all (somewhat increased evacuation), it was obviously only secondarily affected sympathetically through the acid character of the intestinal content, for the absorption was altogether undisturbed.

All authorities, who have studied the diseases of the intestine thoroughly, agree that such a symptom of the intestinal fermental dyspepsia actually exists. Opinions differ only as regards its frequency. My own experience, materially extended in the interval, has taught me that it is one of the most frequent intestinal diseases, particularly if we do not limit the scope of our investigation as was done at first by considering only absolutely uncomplicated cases, but take into consideration the transitions to catarrhal conditions and to gastrogenic diarrheas. For, contrary to the fatty stool in deficiency of the bile, the fermental (*i.e.*, carbohydrate) stool does not remain indifferent to the intestinal mucous membrane; the continual irritation of the acid chyme may lead to catarrhs and inflammations, the products of which (serum, mucus) may efface the characteristic mark of the fermentation of the feces, because of their inclination to putrefaction. On the other hand, it has been proved that the stomach, even if it is quite normal, may still have something to do with the origin of the fermental dyspepsia. On the basis of 17 cases observed recently by *H. Meyer* (106), I must regard the gastric origin probable, at least, for a portion of them and for some of them even proved. The hyperacidity of the stomach content may also lead to fermental dyspepsia as well as the subacidity and achylia. Moreover, as regards the hyperacidity, its connection with the intestinal fermentation is easily intelligible, if we recall that the hyperacidity does not only injure the digestion of starch in the stomach, but, particularly in connection with retarded motility, leads also to fermentations even of the contents of the stomach. Indeed, we could incidentally rediscover the same microbes (long bacilli, yeast) that are known as the stimuli of the gastric fermentations, in the fermenting feces, and thereby add the last link to our chain.

I do not by any means, however, accept the view of *v. Tabora* (74), who speaks of "putrefactive dyspepsia" as the result of achylia, just as "fermental dyspepsia" is the result of hyperacidity. For, as I have said, I have found fermental dyspepsia in cases of achylia even oftener than in cases of increased acidity. Therefore I emphasized above already (VI, A), that the gastrogenic intestinal disturbances do not yield a unified symptom, and I may add here that there doubtless exist cases of fermental dyspepsia which have nothing to do with the stomach, but are of an independent nature. For the present I regard these as the result of the secretory insufficiency of the glands of the small intestine, and I find the confirmation of this in an anatomic study which, under a careful microscopic examination, showed no change, except a very slight—almost doubtful—inflammation of the mucous membrane of the small intestine.

The inferences, that the analysis of the symptom has afforded, are of especial practical interest for the therapy. According to the principle, set down in the discussion of catarrhs (see above), to eliminate from the food those substances which aid the decomposing processes, *in case of fer-*

mental dyspepsia, we shall have to limit, first of all, the carbohydrates in the test-diet, or eliminate them entirely. This procedure actually proves to be here a very suitable and safe one. Since no fermentable matter is secreted into the intestine from the body itself, the fermentation must cease as soon as all carbohydrates have been omitted from the food, and so it does under all circumstances. On a strict meat-fat-diet the patients feel as if suddenly relieved of all complaints, and the excrement becomes solid. If this absolute diet has been followed for a few days, we may begin again to cautiously add carbohydrates, first easily absorbed sugars (milk, cane-sugar), then some fine wheat-flour or starch-foods, zwieback, toast, farina, noodles, rice, and, only after a longer interval, potatoes and vegetables. Potatoes especially seem to be poorly digested by the intestine of persons having fermental dyspepsia, and, therefore, they decompose; this is quite conceivable, for potato-starch is much more difficult to assimilate than wheat-starch (*Strassburger*).

The method which we described here to determine the capacity of the intestine to tolerate carbohydrates under continuous control of the feces, as in diabetes, and to fix the diet accordingly, is more suitable than the one which I chose at first, in which I gradually decreased the carbohydrates of the diet, until the fermentation ceased. This, namely, happens sometimes very late, and the fermentation begins again as soon as more carbohydrates are given. In order to remove the fermental stimulants, water must be entirely cut off, at least for some length of time, and that is brought about best by an absolute albumin-fat-diet strictly conducted for some days. After that we may sometimes suddenly change to a mixed diet, without the recurrence of the disturbance. *The rational diet based on the functional test is here actually victorious.*

In other cases, of course, as said, the fermentation returns readily, as soon as a certain moderate quantity of carbohydrates is exceeded. In such cases other measures must be called to aid. These are determined by the respective condition of the stomach: in case of achylia, hydrochloric acid is given; in hyperacidity, belladonna preparations, bismuth, etc. More important than these is a thorough lavage of the stomach with nitrate of silver (1-3:1000) or with salicylic acid (1-2:1000) after the stomach has been cleaned with water. I have had the best results from it, especially in those forms that had to be considered gastrogenic. As a rule, these cases proved to be less stubborn than those independent cases based on secretory insufficiency of the small intestine. In case of the latter a trial with diastasic preparations is advisable, but I have not sufficient experience with regard to it.

On the whole, it may be said that the fermental dyspepsias and the catarrhs originating in them offer a grateful field. *In the determination of the symptom of fermental dyspepsia, in its explanation and the therapy proceeding from it, the function-test of the intestine has endured its severest test. The practical benefit derived from it, is in itself sufficient to demand its general application. But, besides, we owe it the knowledge of the gastrogenic intestinal disturbances, of the pancreatic achylias, the analysis of many nervous diarrheas and of chronic habitual constipation, all practically important diseases, in regard to which, as we have seen and as we shall yet see, it gives us valuable therapeutic hints.*

B. ABSORPTIVE DISTURBANCES.

Organic and Functional Disorders of Absorption.—Disturbances of Gas-Absorption.

While almost all organic and also most functional diseases of the intestine injure absorption secondarily, isolated independent absorptive disorders of the intestine are very rare occurrences; indeed, we must occupy ourselves very thoroughly with these matters if we would find them. As typical examples of disorders of absorption are usually regarded those digestive disorders, combined with diarrhea, which appear as a consequence of congestion of the intestinal mucosa (weakness of the heart, cirrhosis of the liver), of amyloid degeneration of the intestine, and of diseases of the mesenteric glands *(tabes mesaraica)*. But in all these conditions the picture is seldom simple; conditions of inflammation or ulcerative decomposition of the intestinal mucosa are scarcely ever absent. There are, however, observations of this kind (107) where these objections do not hold, and these prove that when there is exclusively or predominantly a disturbance of absorption, the absorption of fat suffers chiefly, the absorption of albumin less, and the digestion of carbohydrates least, if at all. The amounts of food lost are by no means very great, being far behind the loss observed in affections of the pancreas. Nevertheless the differentiation is certainly not easy in doubtful cases.

Alongside of the organic disorders of absorption, which were cited here only for the sake of comparison, there are also *purely functional* ones, but they are not as prominent and therefore easily overlooked. It is indeed self-evident from the start that the amount of absorption shows variations among different individuals as well as at different times in the same individual, perhaps in the same way as the amount of the daily food ingested, the urinary excretion, etc. As a matter of fact, even with a diet as uniformly composed as the test-diet, the systematic investigation of the feces of healthy persons if extended over greater periods of time, does not at all give the same result constantly, but there are occasionally found somewhat more fat-remains than usual, or other deviations from the normal excrement, which prove the occurrence of such variations. Even more so is this the case in many patients with indefinite digestive disturbances, irregular stool evacuation or other disorders pointing to the intestine, which, like many intestinal catarrhs, as a rule pass from physician to physician under the diagnosis "nervous intestinal trouble." Thus I have often seen that such patients, the condition of whose stool is normal in other respects, regularly excrete surprisingly many and large microscopic remains of muscle-fibers, sometimes even macroscopic fragments of muscle; at other times only the absorption of fat is disturbed. To the latter category of cases *Salomon* (41) made an interesting contribution at the "20. Kongress für innere Medizin." He was able to prove that the disorder could not be ameliorated by administering pankreon (in contrast to the disorders of fat absorption in pancreatic disturbances), and he justly saw in this a valuable aid for differential diagnosis. Evidently many of those "acholic fatty stools without icterus" belong here also. But I will not go further into these intricacies of diagnosis. Therapeutically these cases are, of course, especially satisfactory, because they can easily be ameliorated by the diminution of that part of the food-stuff which is especially badly absorbed.

There is yet to be mentioned here briefly a special kind of absorptive disorder, which undoubtedly occurs as a seemingly independent anomaly, a *diminished absorption of gas on part of the intestinal wall,* the consequences of which (flatulence and tympanites) are mostly very unpleasant for the patients. For the normally functioning intestine it may be taken for granted that by far the largest part of all the gas present in it is removed, not through the anus, but by way of the blood through absorption. How else would the fact be intelligible, that persons in good health can take without any trouble colossal amounts of drinks containing carbonic acid? It is self-evident that flatulence and tympanites may have their basis also in increased gas formation or diminished peristaltic action, but these factors can easily be excluded with certainty. As far as my experience goes, I must say that in the other cases that still remain, a careful investigation of the excrement almost always reveals indications of a catarrh which exists or is passing away. So it is also in the stomach. Any one who complains of distention in the region of the stomach, eructation, and pyrosis, which frequently accompanies it, suffer generally from a "spoiled stomach."

C. MOTOR DISTURBANCES.

Among the various affections generally described as disorders of the motor function of the intestine, there are relatively few which occur as independent disorders; the majority have significance only as the secondary symptom of a disease, as a consequence of other disorders. This holds good, *e.g.,* for all cases of paralysis of the intestine and intestinal colic, and also for the well-known lead colic, which as *Nothnagel* (33) justly emphasizes, is a neurosis of sensation rather than a condition of spasm. Somewhat independent disorders are really only the peristaltic unrest of

Kussmaul (Tormina intestinorum nervosa), the nervous diarrhea and the chronic habitual constipation.

Tormina Intestinorum.

I may presuppose that *tormina intestinorum,* the rotatory movements of the intestinal coils accompanied by loud rumbling and splashing, with ultimately visible peristalsis is well known; but I might, nevertheless, point out again that only when the other investigations, especially those of the stomach and of the feces, have given negative results is it justifiable to regard it as a neurosis. Taking the test-diet as a standard, this occurs but seldom; still it may be that my experience on this point is too limited. *In the more careful investigations of peristaltic unrest we feel the want of a method which would enable us to measure the length of time foods remain in the small intestine.* For it is beyond a doubt that just here where the small intestine is partially or entirely affected, the stool evacuation takes place normally and regularly.

Nervous Diarrhea.

The question of *nervous diarrhea,* too, is still far from settled. That is evident from the fact that it is treated by *Nothnagel* and *Ewald* under the neuroses of motility, by *Boas,* on the other hand, under the neuroses of secretion. The existence of a purely nervous diarrhea, that runs its course without any anatomical lesion of the intestine, is beyond a doubt. It is sufficient to refer to the frequently cited example of the diarrhea produced by fear. Further, every physician has probably seen patients in whom the thought alone that they would have no opportunity for defecation suffices to call forth a movement, or in whom very definite psychic causes lead to evacuation. In most of the cases that belong here the proof that anatomic changes in or on the intestine (cicatrices, catarrhs, adhesions) are absent can hardly be brought; and, relying on the results of functional diagnosis, I have expressed my conviction repeatedly above, to the effect that on careful investigation of the feces the host of nervous diarrheas will shrink to a very small group. Besides, referring to the particles of mucus occasionally found, I have in this connection cited the accompanying abnormal processes of fermentation and putrefaction, which here are but seldom absent, whereas they do not fit in with the symptoms of nervous diarrhea.

In addition to this, if a person with normal digestion suddenly has, as a result of fright, thin stools, we can scarcely explain this without assuming that there takes place at the same time a secretion on the part of the intestinal wall. There are, on the other hand, cases in which the emaciation is disproportionately large in comparison with the intensity of the diarrhea; where therefore the absorption is obviously involved, we come to the conclusion *that a nervous diarrhea, as a consequence of isolated primary motor disturbance, scarcely exists at all;* that here we probably always deal with a combination of various functional disturbances.

I must, however, remark here, that strictly taken, only the result of the investigation of fresh excrement is to be considered, not that of the incubator-test. *If the diarrhea is really of purely nervous origin, the freshly evacuated excrement ought not show any signs of pronounced putrefaction or fermentation, it must have exactly the same appearance as the fresh content of the intestine.* On the other hand, since it may naturally include unabsorbed remains of starch and albumin, it may readily ferment or putrefy in the incubator test.

But even if the stool investigation has often a value inferior to that of the other clinical signs for the diagnosis, since it can be made use of only by exclusion, nevertheless it gives us a valuable suggestion for the treatment; I mean especially the dietetic treatment, for the total therapy must naturally be, first of all, physical or general. When this or that food-stuff is not easily tolerated, especially poorly digested, the composition of the diet must be altered; I would, however, recommend very particularly here, too, as in case of catarrh or fermental dyspepsia, that the course of the processes of decomposition in the feces, kept in the incubator, be taken as the starting point in the choice of diet. *In fermentation a predominantly albuminous diet, in putrefaction a carbohydrate diet, is the rule.* A sudden change of diet is occasionally of surprising efficacy in its results, even, *e.g.*, in the form of a vegetarian regimen. The scientific basis of this procedure is explained by the fact that the flora of the intestinal content requires some time in the case of a sudden change of diet before it can adapt itself to the culture medium [*Lembke* (108)]. During this intermission the irritation caused by the products of decomposition, the formation of which does not tarry long in the cases in question, are lost, and that is of great advantage. Another thing I would always advise is rest in bed. It is beyond a doubt that with certain patients every bodily agitation, even that connected with spontaneous movement, suffices to relax peristalsis, just as, conversely, with others travel on the railroad or on a steamship unfailingly occasions constipation. I attribute some successful results, which at the start were never intended, partly to the rest in bed at the time of the functional examination, which I have always exacted at least for a few days, and partly to the fact that many patients were unaccustomed to the composition of the test-diet. Most vividly I remember, among others, a young man who had from earliest childhood suffered from diarrhea, which had defied all treatment. I explained to him that I would pass judgment on his condition only after investigation by means of the test-diet, and to our greatest surprise, thereupon a normal stool ensued every other day.

Chronic Constipation.

Gastrogenic Constipation.—Spastic and Atonic Constipation.—Nature of Chronic Constipation.—Therapeutics.

Before we proceed to the much discussed chapter of chronic constipation, which has become much more intelligible to us through the functional test, let us consider briefly *the forms of constipation originating in the stomach.*

It has been remarked (Chapter VI, A) that in contrast with achylia where diarrhea is usually accompanied by secondary disturbances of the intestine, hyperacidity is often followed by constipation. This, however, is not always the case. There may be hyperacidity of the stomach combined with diarrhea, as well as achylia with chronic constipation. Although the latter combination is not frequent, I have seen it repeatedly, and have come to the conclusion that in this case the constipation developed gradually from a chronic gastrogenic catarrh of the small intestine *and of the large intestine.* These constipations react remarkably well to hydrochloric acid. More difficult to explain is constipation due to hyperacidity. Some thought that the great disinfective power of the increased hydrochloric acid of the stomach could, perhaps, reduce the growth of the bacteria of the intestines to such an extent that the natural stimuli of the peristalsis issuing from the products of decomposition would be diminished. This would then be the counterpart to the formerly current, but now essentially modified explanation that the gastrogenic diarrhea is caused by the loss of the protection of the gastric disinfectant. What the actual explanation is cannot be stated with certainty. True, *Strassburger* (84) with the aid of his method of weighing the bacteria of the feces found in a relevant case remarkably few bacteria, but as the primary constipation, as we shall see, is also regularly accompanied by a decrease in the number of the bacteria, this does not decide the question. In general, in case of constipation through hyperacidity, the analysis of the feces shows a far-reaching, nay, almost complete correspondence with the conditions in case of genuine constipation (of which we shall speak immediately), so that we cannot grant a separate place to the gastrogenic constipation.

Fleiner's classification of the *chronic habitual constipation* into the *atonic* and the *spastic* forms has received general assent. From a clinical point of view, too, it is perfectly justified, for, while in the former the only symptom of disease is the retarded and more difficult evacuation of the bowels—aside from the purely nervous complaints of dizziness in the head, congestions, feelings of tension in the abdomen, etc.—in the latter the attendant state of irritation or inflammation of the colon, together with cramps, alternating in periods and extent, the mucus discharge and the direct and reflex participation of other abdominal organs are noticeable signs. Considered from a pathogenic standpoint, however, both forms are evidently identical, for spastic constipation is only in very rare cases the result of primary colitis; as a rule the condition is the reverse: the constipation, which in the case of "atonic" patients, may continue for a long time without injury to the intestine, leads in irritable persons to spasms and to inflammation of the colon. At least my investigations, as regards the feces under the test-diet, have revealed no difference between the two forms, unless the characteristic changes, as a rule, become more conspicuous in the atonic constipation than in the spastic.

Briefly, these characteristic changes consist in this, that feces are produced which are not only too hard, but also digested in too small a mass and too thoroughly.

Subjecting a patient with chronic constipation to a test-diet for some time, and gathering his feces of a certain limited period and comparing it with the excrements of a person with healthy intestine, treated in the same manner, we find, as a rule (I base my statement on a great number of individual investigations), not only a decrease in the whole mass and the water-content (see Fig. 14), but also a remarkably small amount of dry substances in the whole fecal mass. Examining it with the microscope, it is noticeable that in the detritus-like mass only extraordinarily small remnants of food substances can be seen, here and there a tiny remnant of a muscle-fiber, no potato-cells at all, few soaps of lime.

In addition to this *Strassburger* (84), who made similar observations, found by means of his method of weighing bacteria, that the entire mass of microbes in the feces of persons suffering from chronic constipation is much lighter than under normal conditions (about ⅔ of the latter). In order to find unobjectionable figures I have induced *Dr. Lohrisch* (109) to make systematic digestive experiments, each one extending over three days' test-diet. The result was, that while normally *an average* of 59.3 Gm. dry excrement with a content of 266.1 calories was evacuated, in constipated persons only 33.9 Gm. with 179.2 calories, was formed. The ratio of the entire nitrogen-content was

2.98:1.55; that of the fats as 13.78:8.36; that of the carbohydrates as 1.8:1.39. *If, however, we produced constipation artificially by means of opium, in persons with healthy intestines, only the amount of water of the feces was reduced, that is to the same amount as in naturally constipated persons, the absolute mass of the dry substance and the amount of N, fat, and carbohydrate remained the same as before.* Particularly interesting and of practical value is the fact affirmed likewise by *Lohrisch,* that the cellulose, too, at least the delicate cellulose of fresh vegetables, is better digested by the digestive canal of persons constipated than otherwise: the corresponding numbers for the test-diet (in which only 190 Gm. boiled potatoes, and 100 Gm. zwieback, of the components, containing cellulose, are administered daily) are: 0.869 Gm. (normal) and 0.684 Gm. (constipation).

Against these results this objection, at most, could be raised, that the small content of food-remains in the feces of constipated persons, is not the consequence of too good an absorption on the part of the intestines, but is due to increased decomposition by the intestinal bacteria. In this case, of course, we would have to suppose that the bacteria in the intestines of constipated persons—since their content is *reduced,* according to *Strassburger's* experiments—display a considerably increased activity. This objection, however, loses its justification if we subject the behavior of the putrefactive and fermentative products of the feces and urine to a critical examination. Though in literature there are various reports to be found that indican excretion is increased in fecal stagnation, they refer exclusively to the urine, and do not concern the feces, which come first into consideration. They would prove, at most, that an increased absorption of indican has taken place, and this is only natural, since the feces of constipated persons remain long in the large intestine and inspissate there. Concerning the theoretically more important ethereal sulphates, the reports differ so greatly that an increased production is improbable.

Much more important, however, is the experience that the fresh feces of constipated persons, contrary to most other feces, shows no signs of increased decomposition. The feces of uncomplicated constipation consists of hard, firm lumps; it is almost odorless, reacts neutrally or almost neutrally, and when examined by means of the incubator-test, shows neither fermentation nor putrefaction. This agrees with the fact that patients suffering from chronic constipation, insofar as secondary symptoms of irritation or inflammation do not appear (hence, especially patients with atonic constipation), are in nowise subject to abundant gas-formation or to other disturbances which could be traced to increased processes of decomposition. On the contrary, such persons, apart from the difficult defecation and the general accessory neurasthenic symptoms, enjoy undisturbed health and an excellent appetite.

The explanation of the facts in the case of chronic constipation is therefore this, that first of all the digestion is too good. The result of this is, that the culture-medium is too poor for development of bacteria, so that the products of decomposition are fewer and the natural stimuli of the peristalsis are decreased, i.e., the motoric action is diminished. Just as in the case of nervous diarrhea, the motoric derangements are also here not primary, but secondary.

Now the constipation of the patient through hyperacidity of the gastric juice becomes intelligible. As is well known, the albuminous substances in case of hyperchylia are very well digested in the stomach already; high HCl values are furthermore strong stimulants for the pancreatic juice, and it need not surprise us that the food is entirely too well digested under such circumstances.

The question arises whether the new recognition of the pathogenesis of chronic constipation, which we owe to the function-test, is able to produce *results as regards therapeutics?*

If the first cause of the constipation is a too good digestion of the food, one would think that it is an easy matter to cure the evil, by bringing about an artificial disturbance of the digestion. But closer examination will teach us that it is not as simple as it seems to be, for we know that some constipated persons are entirely unaffected by large doses of purgatives which would cause very copious diarrheas if taken by healthy people. *Roos* (110), and later *Strasburger* (111), have made experiments through artificial introduction of cultures of yeast and bacillus coli communis to increase the processes of decomposition in the intestine and thereby to improve peristalsis. The experiments failed utterly, *Strasburger* could not even produce thereby an increase of intestinal bacteria. That is natural, for if in reality the better digestion is primary, and the degree of bacterial settlement is only an incident of the remains of the food-stuffs, the lever must be put to the digestion itself, the attempt must be made to deteriorate it, in order that the processes of decomposition may take place in a wider field. In fact, we are working for a long time more or less consciously in this direction.

It is an old custom to prescribe for constipated persons a coarse diet containing abundant waste matter: black bread, salads, fruit, nuts, etc., evidently in order to disturb the digestion. These foods, rich in cellulose, are poorly digested by the healthy intestine, as the experiments of *Voit* and his disciples have taught us: these poorly digestible cellular membranes protect the enclosed starch granules and albumin substances from the influence of the digestive juices, and deliver them to the decomposing bacteria, which form out of them products of cleavage (volatile fatty acids, gases, etc.) which stimulate the intestine: the stool becomes massy, pappy, and is oftener discharged. *In case of constipated persons, however, we often find that the stool, notwithstanding such a diet, remains hard, and the evacuation stops after it just as before.* This is explained by the fact, mentioned already, that the intestine of constipated persons digests cellulose better than the normal intestine; while we can detect remains of carrots, of green vegetables, and even of potatoes in the stool of healthy persons even with an unaided eye, we look for them in vain in the feces of a constipated person. It is yet unknown in what manner the intestine of these patients dissolves the cellulose. It is improbable that this is done by the intestinal bacteria alone, as they are scantier in the intestine of constipated persons than otherwise. But here we shall not enter deeper upon this question.

A diet rich in waste-matter will therefore not serve our purpose—aside from the fact that it cannot be expected of everybody; we must try to add such components to the food as cannot be dissolved. As such woody or corky cellulose (bran, pieces of cork, saw-dust, etc.) or sand come into consideration. All these things have been tried for constipation, but without material result, and this is intelligible if we consider that they are surely apt to make the feces more voluminous, but not softer or more abundant in water; and just this is the point to be attained if the evacuation is to be facilitated.

A substance which fulfills both conditions (greater volume and increased quantity of water of the feces) and *which, at the same time, possesses the advantage of not being injurious and irritating, is the bacteriologically well known agar-agar.* Agar-agar consists mostly of hemicellulose; the amount of cellulose it contains is, according to our experiments, only 0.6 per cent. In a soaked condition it gives up its water only with difficulty, not even under the influence of putrefaction, which it resists for a long time. For bacteria, as is well known, do not transform agar-nutrition to a fluid. As regards the conduct of agar-agar in the digestive canal, I have performed many experiments which show the following results: if we administer agar-agar in the current form of a stripe about 50 Cm. long, and of the thickness of a straw up to that of a finger, cut into small pieces, it swells in the mouth, more so in the stomach, and reappears in the same condition unchanged in the feces. The feces become thereby more watery and softer; its dry content increases with the quantity of the dry agar taken in (112) (see Fig. 15). Agar, taken in this manner, exerts no irritating influence on the digestive organs, not even in large doses, only when administered in the form of fine powder did I notice occasional pain in the bowels and diarrhea, which I am inclined to regard as the effect of a more rapid and larger swelling. My expectations concerning the administration of agar-agar (in small pieces) to the constipated, have been completely realized. In case of constipation, also, the swollen agar passes the digestive canal unchanged and renders the feces—if given in proportionate quantity—voluminous and soft. Indeed, it may under circumstances unduly impede, as undigested ballast, the absorption of other food-components, particularly of those that contain cellulose. But it does not directly cause, at least not regularly, the spontaneous evacuation of the loosened fecal mass. This is due to the fact that agar does not, at the same time, cause irritation in the intestinal walls, as do the processes of decomposition, which are normally formed in the large intestine. As we have seen, these are absent in chronic constipation or are not formed in a sufficient mass, and in this respect agar does not change matters. Here, again, we have another clear evidence that in addition to the mechanic item (of accumulation of the feces in the descending colon and in the rectum) there must also be a chemic one, in order to bring about an evacuation of the feces, and that the latter is particularly necessary when it is difficult to stimulate the intestinal walls because of the chronic constipation. In order to supply this missing chemical stimulant, I have decided, after many experiments, to add to the agar-agar a small quantity of extract of cascara (15-25%). According to the process worked out by the chemical laboratory of Helfenberg A.-G., this may be combined with the dry agar in such a manner that it will not give it any taste, and, as was shown by special experiments, will, for the most part, diffuse from the swollen agar in the intestine alone. This addition of cascara is not supposed to act as a purgative—for this the additional quantity is much too small[1]—but to supply the missing stimuli of the natural processes of decomposition in the intestinal content, and thus bring it about that the daily portion of agar is reduced to a smaller quantity.

The cascara-agar, which is thus improved, is introduced into the market by the chemical laboratory of Helfenberg A.-G. under the name of *"Regulin,"* and *I have been able, indeed, to produce very good results with it.* The patients take this prescription, which is not at all disagreeable, best if administered with apple-marmalade or some similar compôte, also with potato-broth, from a teaspoon to two tablespoons, daily, which amounts to about 1½-8 Gm. Since this prescription is not to be regarded as a purgative, but serves, as its name signifies, to regulate the evacuation, it must be taken persistently every day. According to its nature it is especially indicated in the light and moderately severe cases of both atonic and spastic forms. In severe or in inveterate cases it is sometimes necessary in the beginning to make use of other methods too, be they actual purgatives or physical measures (massage, electricity, hydrotherapeutics, enemas). As a rule, after continued use of regulin in time spontaneous evacuations will occur. Habituation to the drug does not occur; what is more, the dose may in most cases later be diminished. Diarrhea never occurs, not even after a very large dose. Hence it may be given to children without hesitation.

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[1] The watery cascara extract does not stimulate the intestine as much as does the alcoholic.

The *rational basis of the regulin therapeutics* is supported by the confirmation and endorsement of a number of authors (113) and the extended use of it among the general public, apart from my own wide practical experience. While at first I endeavored to develop it further through combined treatment with substances of similar effect, especially with liquid petrolatum ("pararegulin"); I desisted from it later. If one carefully analyzes the condition of each individual case by means of an exact examination of the whole digestion with the aid of stomach-tube, test-diet and rectoscope, it will be clear what measures are to be taken alongside with the regulin. These measures concern not only the physical methods already mentioned and the purgatives, which in some cases cannot be entirely eliminated, but extend naturally to the diet, and under certain conditions to the whole manner of living. It leads us too far away to discuss details here. Only on two points I wish to touch briefly.

The one concerns the enemata, especially the oil enema. In general I am opposed to the enema therapeutics, particularly in the spastic form. I have noticed too many irritations from its effect. But I admit that in the case of atonic constipation and in temporary constipations of healthy persons, mild enemata in the form of lukewarm water and in small quantities (½ liter) are harmless and often efficacious. But, at least, the so-called "high" and "large" enemata should be stopped. The contention that we reach the ileocecal valve is not tenable, for that occurs only in exceptional cases. Therefore it is useless to push the intestinal tube farther than just above the anal sphincter. I think it is harmful to use medicated enemata for constipation, except Seltzer-water enemata which, I noticed occasionally, have a refreshing effect on the large intestine. It cannot be disputed that the *Kussmaul-Heiner* oil-enema sometimes bring about good results; but it is wrong to reason that they "soften" the hard fecal balls. The chief effect of the oil-enema is certainly due to the mildly stimulating action of the fatty acids split up by the bacterial decomposition. Sensitive intestines, however, may suffer even through this. Besides the oil diminishes friction. I certainly cannot agree to look upon the oil-enemata as a panacea for all kinds of constipation and for all the different catarrhs of the large intestine, as does *Ebstein*, who developed them into a systematic treatment.

The second point is concerned with *rest in bed* as an aid in the treatment of constipation. Generally it is said that the constipated persons should move about very much, but there are many people who, in spite of very much activity in their calling, display a high degree of constipation. Indeed, at least passive motion in many respects tends to retard stool (riding, traveling on trains). Under such circumstances rest in bed in connection with a diet rich in fat (test-diet with the addition of milk or butter) often shows remarkable results. I do not therefore neglect to have the patients confined to bed during the function-test, which in this respect I use also as a time for observation. Moreover, I am not alone in my experience. *Boas* (114) also cites cases of this kind. Obviously, whereas the agitation of the intestines through walking stimulates peristalsis in those disposed to diarrhea, it serves in others as an inhibitory stimulus. As long as we have not entered deeper into the complicated condition of the innervation of the musculature of the large intestine, we must, unfortunately rest content with such vague explanations.

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